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TECHNICAL SUPPORT FOR
ROCKY MOUNTAIN ARSENAL
10-25-90

DRAFT FINAL
RISK ASSESSMENT
LIME SETTLING BASINS
VERSION 2.1

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Prepared by:

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Prepared for:

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EXECUTIVE SUMMARY

The preferred alternative Lime Settling Basins (LSB) Interim Response Action (IRA) selected to inhibit further migration of contaminants from the LSB includes moving the stockpiled lime sludge adjacent to the LSB back into the LSB, the construction of a subsurface barrier (i.e., slurry wall), placement of a soil cap and vegetative cover, and the installation of a groundwater extraction system. This IRA is expected to be completed in approximately 6 months. The LSB IRA Risk Assessment (RA) presents the methodologies, quantitative and qualitative results, and assumptions used to determine if a potential risk exists to humans and biota from the activities of the LSB IRA.

The activities associated with the relocation of the lime sludge into the LSB and the installation of the slurry wall are the focus of this RA, since they include disturbance of soils identified as contaminated. Twenty-five contaminants of concern (COCs) were selected for evaluation in this RA based on the data provided in the remedial investigation at Rocky Mountain Arsenal. The populations evaluated for health risks from the LSB IRA are on- and off-post human populations and on-post biota populations. In the human health evaluation inhalation of vapors and suspended particles were identified as the critical exposure routes. A conservative Gaussian plume air dispersion model was used to estimate ground-level air contaminant concentrations for the COCs at the point of the nearest on-post and off-post populations. Contaminant intake rates were calculated using the concentrations predicted from the air dispersion model and subsequently were compared to toxicological data to determine carcinogenic and noncarcinogenic human health risks. Conservative exposure assumptions were used consistent with United States Environmental Protection Agency (EPA) risk assessment guidance.

Risk evaluations for human health indicate an increased risk of cancer of 1.7×10^{-5} (i.e., approximately 2 chances in one hundred thousand of contracting cancer) for on-post receptors and 1.0×10^{-6} (i.e., approximately 1 chance in one million of contracting cancer) for off-post receptors. Noncarcinogenic hazard indices were less than a reference level of one for on- and off-post receptors, indicating noncarcinogenic health effects are not likely from LSB

IRA activities. The potential for noncarcinogenic effects may be underestimated since dose response estimates for the inhalation route were available for only 3 of the 25 COCs evaluated.

Assessment of impacts to ecological receptors was qualitative because information on media intake rates and chemical-specific allowable exposure rates for biota are not generally available. However, given the limited area of disturbance and the short duration of the LSB IRA activities, it was concluded that ecological receptors will not be adversely affected by the implementation of the LSB IRA.

Social impacts from IRA activities were also evaluated in this assessment. In consideration of the remote location of the LSB and the nature of IRA activities, adverse social impacts such as increased noise levels, emission of offensive odors, decreased visibility, and increased vehicular traffic are not expected.

1.0 INTRODUCTION

This document represents the results of a risk assessment (RA) that was performed to assess risks from the Interim Response Action (IRA) activities that are being considered for the Lime Settling Basins (LSB) in Section 36 of the Rocky Mountain Arsenal (RMA). The RA is being performed in response to the following litigation efforts.

On February 1, 1988, a proposed Consent Decree was lodged in the case of United States v. Shell Oil Company with the U.S. District Court in Denver, Colorado. The proposed Consent Decree was revised after public comments were received, and a modified proposed Consent Decree was lodged with the Court on June 7, 1988. In February 1989, a Federal Facility Agreement (FFA) was entered into between Shell Oil Company and five federal agencies: the U.S. Environmental Protection Agency (EPA), the U.S. Army (Army), the Department of the Interior (DOI), the Department of Health and Human Services (DHHS), and the Department of Justice (DOJ). Established procedures for implementing the RMA cleanup program as specified in the Technical Program Plan, and incorporated many provisions of the modified proposed Consent Decree. The Army and Shell Oil Company agreed to share certain costs of the remediation to be developed and performed under the oversight of the EPA, with opportunities for participation by the State of Colorado.

Final remediation at RMA will be a complex task that will take several years to complete. The FFA specifies 13 IRAs which will be implemented until such a time that a final remedial strategy has been selected. One of the 13 IRAs specified in the FFA is "Remediation of Other Contamination Sources". The LSB constitutes one of several sites being addressed by the IRA for Remediation of Other Contamination Sources.

1.1 BACKGROUND AND RATIONALE

The Shell Oil Company, the EPA, and the Army have agreed to conduct IRAs for the remediation of contamination sources (i.e., "hot spots") on the RMA. These IRAs are intended to mitigate continuing migration of contaminants from such sources and to minimize potential exposure of human and ecological receptors until a final remedial response action for RMA is implemented.

- Evaluation of the uncertainty in the analyses.

The ecological RA is based on procedures specified in the EPA Region I Supplemental Risk Assessment Guidance for the Superfund Program - Part II (EPA, 1989a). This RA is qualitative in nature because information on media intake rates and chemical-specific allowable exposure rates that would permit the quantification of risk to ecological receptors were not readily available. Potential risks to biota were evaluated based on the following steps adapted from the ecological RA guidance document referenced above:

- Identification of COCs
- Identification of contaminant transport pathways
- Development of toxicological profiles for COCs
- Characterization of potentially exposed populations and habitats and applicable exposure pathways
- Discussion of the potential for unacceptable exposures
- Identification and discussion of the major assumptions used in the assessment together with other sources of uncertainty.

1.3 REPORT FORMAT

Section 2.0 contains a brief description and history of RMA and the LSB. A description of the preferred alternative IRA selected for the LSB IRA is given in Section 3.0. COCs for the human and ecological RA are identified in Section 4.0. The human health RA is found in Section 5.0, while the ecological RA is contained in Section 6.0. Section 7.0 discusses social, physical, and aesthetic impacts, such as noise, odor, visual, and vehicular impacts that may occur as a result of LSB IRA activities. Section 8.0 presents the conclusions and recommendations based on the results of the human health and ecological RA. Section 9.0 contains references cited in the report. The methodological approach and results of the preliminary human health screening-level RA are presented in Appendix A. Toxicity profiles for the COCs which were developed as part of the human health and ecological risk assessments are included in Appendix B.

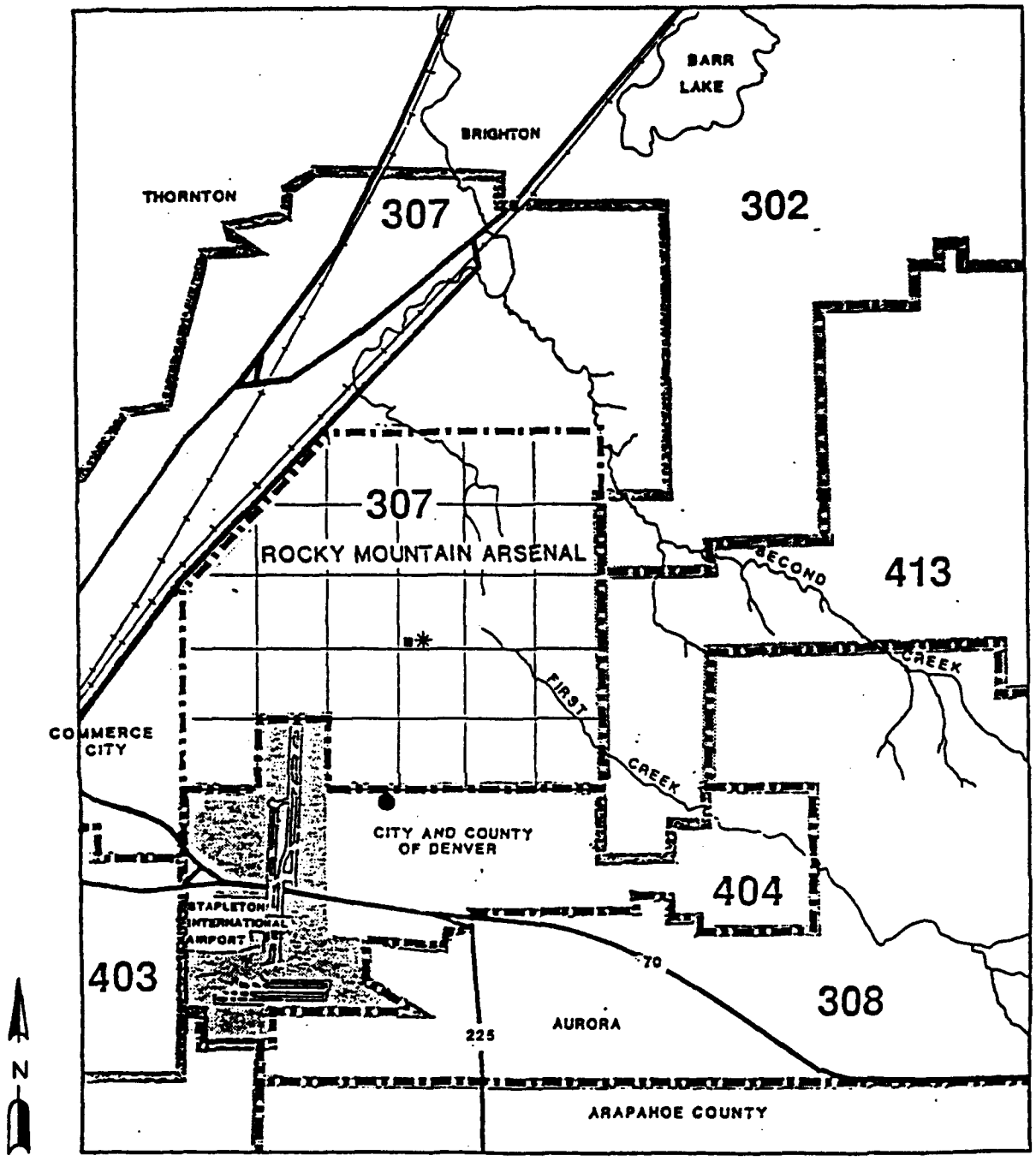
2.0 SITE DESCRIPTION AND HISTORY

RMA occupies approximately 27 square miles in Adams County, directly northeast of metropolitan Denver, Colorado (Figure 2-1). The property was purchased by the U.S. Government in 1942 for use in World War II to manufacture and assemble chemical warfare materials, such as mustard, lewisite, and incendiary munitions. Starting in the 1950s, RMA produced the nerve agent isopropyl methylphosphonofluoridate (GB) until late 1969. A significant amount of chemical warfare material demilitarization took place during the 1950s and 1960s. After 1970, RMA was primarily involved with the demilitarization of chemical warfare materials. The last military operations at RMA ended in the early 1980s. In addition to these military activities, major portions of the plant facilities were leased to private industries, including Shell Oil Company, for the manufacture of various insecticides and herbicides, between 1947 and 1982. In November 1988, the RMA was reduced to inactive military status with the remaining mission at RMA for contamination cleanup.

During the 1940s and 1950s, wastewater from the production of Army agents was routinely treated prior to discharge in unlined evaporation ponds. This treatment involved the addition of lime to the wastewater to precipitate metals. Wastewaters produced in the South Plants were channeled through the LSB prior to gravity discharge to Basin A. The lime sludge produced by the precipitation process contained elevated levels of heavy metals, arsenic, and mercury. Subsequent discharges of pesticide production wastewater resulted in the addition of pesticides to the LSB sludge.

The areal extent of the LSB for this IRA includes the three settling basins located in the southwest corner of Section 36, and the surrounding 360-degree area extending out to the predetermined North Central Study Area Report-site boundary. A map of the LSB in relation to surrounding areas is presented in Figure 2-1. The LSB occupy about five acres.

A number of studies have been completed to characterize the nature and extent of contamination in the soil, sludge, and groundwater in the vicinity of the LSB (ESE 1987, ESE 1988; EBASCO 1989; WCC 1989). Results of these studies confirm the presence of the chemicals expected as a result of the historical activities associated with the LSB. The soil



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U.S. Army Program Manager
Rocky Mountain Arsenal Contamination Cleanup

Figure 2-1
Location of Contaminant Source and Nearest Off-post Residence

3.0 PREFERRED ALTERNATIVE INTERIM ACTION TECHNOLOGY

The preferred alternative IRA for the LSB is construction of a 360-degree subsurface barrier (i.e., slurry wall), and the placement of a soil cap and vegetative cover. The barrier will completely surround the LSB and will be anchored a minimum of five feet into the Denver Formation. Because the Denver Formation is relatively impermeable in this area, anchoring the barrier into the Denver Formation will inhibit lateral contaminant migration. Prior to barrier construction, lime sludge, currently stockpiled in areas adjacent to the basins, will be relocated back into the settling basins. Soils excavated during construction will also be placed within the boundaries of the barrier. Relocation of lime sludge into the basins will require approximately 60 days to complete followed by an additional 120 days for barrier construction. At the completion of the barrier construction, a soil cap and vegetative cover will then be placed over the LSB. The cover will be sloped from the center to facilitate runoff. The cover will inhibit downward migration of contaminants to the groundwater through surface infiltration.

A groundwater extraction trench or well will be constructed within the subsurface barrier. Groundwater will be periodically extracted from the trench or well to maintain an inward hydraulic gradient across the barrier. This will help limit the migration of contaminated alluvial groundwater across the barrier, and increase the efficiency of the barrier. Extracted groundwater will be treated to remove organic and inorganic contaminants. Treatment will be performed either at a CERCLA Wastewater Treatment System or at a separate treatment facility implemented and operated for this IRA.

4.0 CONTAMINANTS OF CONCERN

The COCs were selected from analytical soil data present from remedial investigation studies at RMA. Analytical data reported in the RMA Remedial Investigation (RI) Report (ESE, 1987; ESE, 1988) and the North Central Study Area Report (Ebasco, 1989) were reviewed to characterize the nature and extent of contamination in the area where the LSB IRA activities will occur. LSB IRA activities will predominantly entail displacement of soils. Volatilization of contaminants from groundwater exposed during construction of the slurry wall is expected to be minimal based upon the construction method of backfilling with slurry as the excavation proceeds, which does not expose an open trench for any extended length of time. On-site air monitoring data were not used in this RA, since the data probably represent multiple source emissions, and cannot be correlated with LSB contaminants.

Twenty-five contaminants were detected in soils at the LSB and considered COCs for the human health and ecological RA. Table 4-1 identifies the COCs and presents their average and maximum concentrations and the number of detections measured in soil. For quantitative evaluation, average contaminant concentrations were computed as the upper 95th percentile of the arithmetic mean, consistent with the reasonable maximum exposure (RME) approach recommended by the EPA (EPA, 1989a). In addition, contaminants reported in samples as below the detection limit were conservatively assumed to be present at one-half the detection limit and were included in calculation of the mean concentration (EPA, 1989a). Furthermore, estimated concentrations (i.e., those reported as less than a given value) were assumed to be present at the given concentration. The average RME concentrations used in this assessment to predict risks are viewed as a conservative representation of the average concentrations throughout the site.

Table 4-1 Mean and Maximum Soil Concentrations of Contaminants Present at the Lime Settling Basins

	Mean Concentration (µg/g)[1/]	N[2/]	Maximum Concentration (µg/g)	Boring (Phase)[3/]
Organics				
Aldrin	163	70	600	3172 (I)
Benzene	4.97	12	6.00	3176 (I)
Chlordane	156	70	560	3421 (II)
Chlorobenzene	1.31	12	2.00	3167 (I)
Chloroform	6.52	12	7.00	3167/3171 (I)
Chlorophenylmethyl sulfide	7.38	70	20.0	3168 (I)
Chlorophenylmethyl sulfoxide	12.3	70	23.0	3421 (II)
Chlorophenylmethyl sulfone	18.0	70	50.0	3168 (I)
Dieldrin	51.3	70	120	3413 (II)
DDE	6.86	70	25.0	3413 (II)
DDT	1.93	70	7.00	3171 (I)
Dibromochloropropane	0.203	70	0.500	3421 (II)
Dicyclopentadiene	4.34	70	7.10	3421 (II)
Endrin	11.0	70	36.0	3413 (II)
Fluoroacetic acid	221	7	260	3422 (II)
Hexachlorocyclopentadiene	0.306	48	0.500	3413 (II)
Isodrin	80.0	70	300	3166 (I)
Methylene chloride	1.52	11	2	3164 (I)
Tetrachloroethene	0.191	10	0.250	3422 (II)
Metals				
Arsenic	166	70	370	3730 (II)
Cadmium	1.71	70	3.70	3167 (I)
Copper	76.2	70	270	3168 (I)
Lead	71.3	70	230	3168 (I)
Mercury	29.0	70	110	3421 (II)
Zinc	167	70	500	3168 (I)

1/ Mean concentration was computed as the 95th percentile upper confidence limit of the arithmetic mean (EPA, 1989a). Samples reported as below detection limits were assumed to be present at one-half of the detection limit. In addition, estimated concentrations (i.e., those designated > or <) were assumed to be present at the given concentration and were included in calculation of the mean.

2/ Indicates the number of samples used to calculate the mean, including those assumed to be one-half the detection limit.

3/ Indicates either Phase I or Phase II sampling (ESE, 1987; ESE, 1988).

5.0 HUMAN HEALTH RISK ASSESSMENT

The human health RA developed in this section includes a quantitative assessment of contaminant exposures, a summary of the toxicological data for each contaminant, a characterization of the potential risks associated with exposures at the site and a discussion of the uncertainties associated with this approach. Each step of this approach is presented in relation to the LSB IRA in the sections which follow.

5.1 EXPOSURE ASSESSMENT

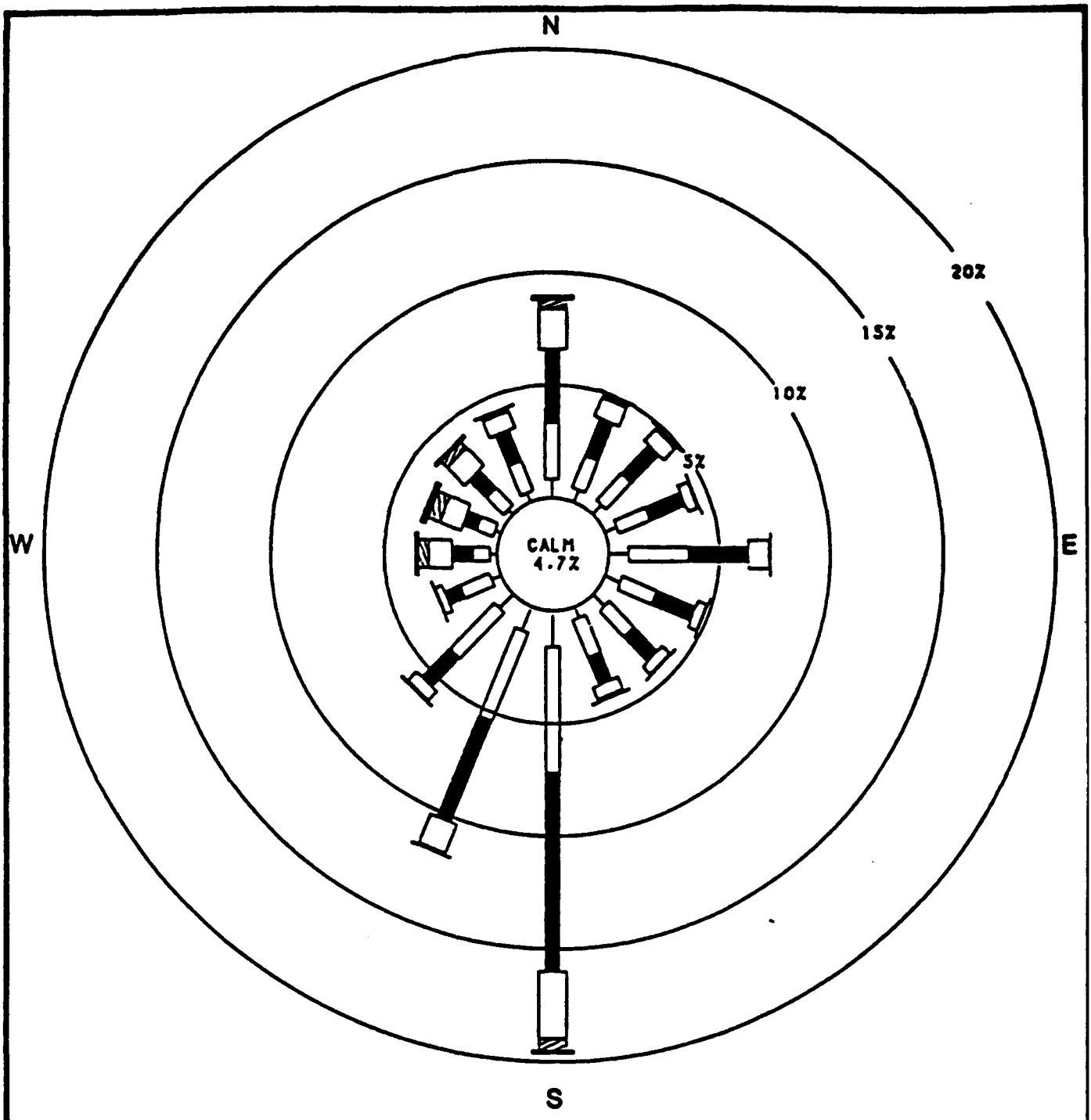
The objective of the exposure assessment is to estimate the type and magnitude of exposures to the chemicals of potential concern that are present at or migrating from the LSB. The results of the exposure assessment are combined with chemical-specific toxicity information to characterize potential risks.

The principal components of the exposure assessment include (1) identification of potentially exposed populations, (2) identification of potential exposure pathways, (3) quantification of exposure point concentrations, and (4) quantification of chemical intakes. These specific components of the exposure assessment are discussed below.

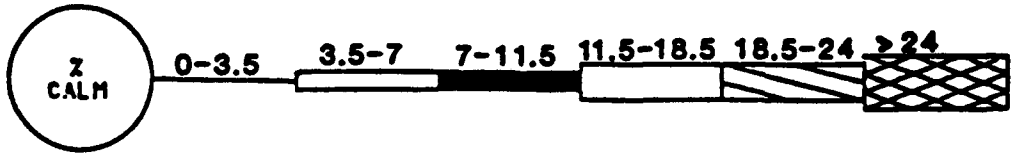
5.1.1 Identification of Exposed Populations

Actual exposures of on- and off-post human populations to contaminants transported by air depend on wind direction, wind speed, and the size of wind entrained particulates. Wind direction and speed data for Stapleton International Airport, southwest of RMA, are depicted in the wind rose diagram presented in Figure 5-1. As indicated in the figure, winds from the south are most frequent, although winds are observed in all other directions. Wind speeds are generally highest from the north, south, and west, and are generally below 24 miles per hour. Due to the high variability in wind direction and speed, all identified populations surrounding RMA may potentially be exposed to contaminants via air transport from the LSB IRA activities.

The closest on-post population to the LSB is the RMA Fire Department which is approximately 225 meters southwest of the basins (see Figure 2-1).



WIND SPEED CLASSES (MPH)



Source: ESE, 1988a

Prepared for:

U.S. Army Program Manager
Rocky Mountain Arsenal Contamination Cleanup

FIGURE 5-1

Annual Wind Rose for RMA Vicinity
(Stapleton International Airport 1975-1979)

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated

The closest off-post population is located approximately 2 miles due south of the excavation, in an area zoned for residential and light industrial land uses. This area includes residential neighborhoods such as Montbello and light industrial zones in and around Stapleton International Airport.

5.1.1.1 Population Distribution for On-Post Area

RMA is an active post with a daily working population. This working population is distributed predominantly in Sections 1, 2, 3, 4, 35, and 36. The closest population that may be potentially exposed to LSB IRA contaminants are the RMA Fire Department employees. The RMA Fire Department is located in Section 36 approximately 225 meters southwest of the LSB.

5.1.1.2 Population Distribution for Off-Post Area

Data used to characterize human populations in the area surrounding RMA were obtained from the Denver Regional Council of Government Population and Employment Forecasts, as presented in the Exposure Assessment for RMA (EBASCO, 1990). These data are presented for the areas around RMA as Regional Statistical Areas (RSAs). 1990 population and employment estimates for these RSAs are presently unavailable; therefore, estimates are presented for 1985. The population density, employment populations, land use, and an approximate minimum distance to invasive activities undertaken as part of this IRA are presented for each RSA. Population density for each RSA around RMA (RSAs 302, 307, 403, 404, and 413) was estimated by dividing population estimates by the approximate area of each RSA. Because RSA 307 includes RMA, the area of RMA has been subtracted from the total RSA area for the determination of population density.

A small portion of RSA 302, known as the Adams-North Central RSA, borders RMA on the northeast. It is comprised of open space and agricultural lands. Its reported population density for 1985 was approximately 175 persons per square mile. Employment population for this RSA was 4,682 persons in 1985. The minimum distance from the construction site of the LSB to the border of this RSA is roughly three miles.

RSA 307, known as the Adams-South Central RSA, corresponds to Commerce City's planning area located on the north, northwest, and western borders of RMA. This RSA includes Commerce City industrial and residential zones, and had a reported population density of 680 persons per square mile in 1985. The employment population for this RSA was estimated at 34,900 persons in 1985. The closest boundary of RSA 307 to the LSB is roughly three miles.

RSA 403, known as the Denver Park Hill RSA, does not actually share a border with RMA, but is located to the southwest and includes residential areas within Denver city limits. The population density was estimated at 4,725 persons per square mile in 1985. The employment population of this RSA was estimated at 17,200 persons in 1985. The minimum distance to the LSB is roughly four miles.

RSA 404, known as the Denver-Northeast RSA, borders RMA to the south and southeast. This RSA includes residential neighborhoods such as Montbello and light industrial zones in and around Stapleton International Airport. The population density estimate was 1,110 persons per square mile for 1985. Employment population estimates for 1985 indicate that roughly 32,000 persons were employed in this RSA. Of this estimate, Stapleton International Airport employs roughly 18,000 employees. The minimum distance from the RSA 404 border to the area of the LSB is approximately two miles.

RSA 413, known as the Denver-New Airport RSA, includes areas designated as open space and a small portion of this RSA borders RMA to the east. The population density estimated for this RSA in 1985 was 57 persons per square mile, and 190 persons were employed in this RSA in 1985. The distance from the area of the LSB to the nearest border of RSA 413 is roughly three miles.

5.1.2 Exposure Pathways

LSB IRA activities will predominantly involve the displacement of soils and sludges. The primary exposure pathways which contribute to the greatest overall site risk associated with the LSB IRA activities are inhalation of contaminants volatilized from soil media and

inhalation of contaminants sorbed to suspended particulates. Soil ingestion is expected to contribute very little to site risks because the larger particle size required to be cleared by the respiratory tract and ingested are not easily wind entrained to reach receptors and the assumption that the ingestion of particulate matter would be controlled by personal hygiene practices. Contaminated groundwater will be exposed to the air during construction of the slurry wall, although it was determined, using worst-case assumptions, that volatilization of groundwater contaminants will have a minimal impact on risks. Based on the construction method of backfilling with slurry as the excavation proceeds, which does not expose an open trench for any extended length of time, the groundwater contribution would be further reduced.

5.1.3 Estimation of Exposure Point Contaminant Concentrations

Downwind contaminant concentrations in air at the RMA Fire Department and at the nearest RMA boundary resulting from excavation activities were calculated based on soil contaminant concentrations, estimated volatile and fugitive dust emissions, and subsequent atmospheric dispersion. The procedures used to estimate downwind contaminant air concentrations are presented in the following sections.

5.1.3.1 Emission Rate Calculations

Emissions of contaminants from the LSB IRA activities are expected to occur as volatile releases and fugitive dust. Each type of emission was calculated separately as discussed below.

Volatile Emission Rate

The rate of emission of volatilized contaminants from soil was estimated using a landfill equation for volatile releases (EPA, 1988a). The landfill equation is based on Fick's Law of steady state diffusion and assumes that diffusion into the atmosphere occurs at a plane surface where contaminant concentrations remain constant. Diffusion of the contaminants through a soil cover is the controlling factor. Since the LSB are not technically landfills, a clean soil cover thickness (d_{sc}) of 1.0 cm was assumed. The landfill equation and input parameters are presented below:

$$E_{vi} = D_i * C_{si} * A * (P_t^{4/3}) * \frac{M_i}{d_{sc}} \quad (1)$$

where

E_{vi} = volatile emission rate of contaminant i (g/s);

D_i = diffusion coefficient of contaminant i in air (cm^2/s);

C_{si} = saturation vapor concentration of contaminant i (g/cm^3);

A = exposed area (cm^2);

P_t = total soil porosity (0.526 dimensionless);

M_i = mole fraction of contaminant in waste (g-mole/g-mole);

d_{sc} = effective depth of clean soil cover (cm).

The saturation vapor concentration, C_{si}, must be calculated separately for inclusion in Equation (1).

$$C_{si} = \frac{P_i * MW_i}{R * T} \quad (2)$$

where

C_{si} = saturation vapor concentration of contaminant i (g/cm^3);

P_i = vapor pressure of contaminant i (mm Hg);

MW_i = molecular weight of contaminant i (g/mole);

R = molar gas constant (62,361 mm Hg cm^3/mole °K);

T = absolute temperature (K).

Fugitive Dust Emission Rate from Wind

The emission rates of contaminants sorbed to particulate matter were computed separately for wind erosion and excavation activities.

The emission rates due to wind erosion were estimated using the storage pile emission factor equation (EPA, 1989b). The wind erosion emission rate for each COC is the product of the total particulate loading rate, the total area of potentially exposed contaminated soil, and the

soil concentration of the COC. Mathematically, the wind erosion emission rate may be expressed as follows:

$$E_{wi} = E_p * A * C_i \quad (3)$$

where

E_{wi} = wind erosion emission rate for contaminant i (g/s);

E_p = total particulate loading rate per unit area $\frac{kg}{hectare\text{-}hour}$;

A = area of exposed soil (cm^2);

C_i = concentration of contaminant i in soil (µg/g).

Appropriate conversion factors must be applied for consistency in the units.

To compute E_{wi}, the total particulate loading rate E_p must be derived as shown below:

$$E_p = 1.8 * U \quad (4)$$

where

U = maximum 1-hour wind speed (m/s), 11 m/s was assumed;

1.8 = empirical constant $\frac{s - kg}{m\text{-}hectare\text{-}hour}$.

The emission rates for volatiles and wind erosion are combined in Table 5-1 because when used in the atmospheric dispersion model they are modeled using the same area which results in same dispersion coefficient for both volatile and wind erosion emissions. Therefore, the emissions were combined.

Fugitive Dust Emission Rate From Excavation

Fugitive dust emission rates due to agitation of soils during excavation activities were estimated using an equation for unloading a dump truck or backhoe bucket (EPA, 1989b).

Table 5-1 Estimated Contaminant Emission Rates (g/sec)

CHEMICAL	VOLATILE EMISSION MEAN	VOLATILE EMISSION MAX	WIND EROSION MEAN	WIND EROSION MAX	EXCAVATION MEAN	EXCAVATION MAX	EMISSION RATE (V+W)(1) MEAN	EMISSION RATE (V+W)(1) MAX
ALDRIN	3.6E-05	1.3E-04	1.8E-03	6.7E-03	1.7E-05	6.2E-05	9.1E-08	3.4E-07
BENZENE	8.8E-01	1.1E+00	5.5E-05	6.7E-05	5.1E-07	6.2E-07	4.4E-05	5.3E-05
CHLORDANE	7.6E-06	2.7E-05	1.7E-03	6.2E-03	1.6E-05	5.8E-05	8.7E-08	3.1E-07
CHLOROBENZENE	3.4E-02	5.2E-02	1.5E-05	2.2E-05	1.3E-07	2.1E-07	1.7E-06	2.6E-06
CHLOROFORM	2.9E+00	3.1E+00	7.3E-05	7.8E-05	6.7E-07	7.2E-07	1.4E-04	1.5E-04
P-CHLOROPHENYLMETHYL SULFIDE	1.1E-03	2.9E-03	8.2E-05	2.2E-04	7.6E-07	2.1E-06	5.7E-08	1.5E-07
P-CHLOROPHENYLMETHYL SULFONE	3.0E-03	5.6E-03	1.4E-04	2.6E-04	1.3E-06	2.4E-06	1.5E-07	2.9E-07
P-CHLOROPHENYLMETHYL SULFOXIDE	1.8E-04	5.0E-04	2.0E-04	5.6E-04	1.9E-06	5.2E-06	1.9E-08	5.2E-08
DDE	1.8E-07	6.6E-07	7.6E-05	2.8E-04	7.1E-07	2.6E-06	3.8E-09	1.4E-08
DDT	4.7E-08	1.7E-07	2.1E-05	7.8E-05	2.0E-07	7.2E-07	1.1E-09	3.9E-09
DIBROMOCHLOROPROPANE	8.4E-04	2.1E-03	2.3E-06	5.6E-06	2.1E-08	5.2E-08	4.2E-08	1.0E-07
DICYCLOPENTADIENE	2.1E-02	3.4E-02	4.8E-05	7.9E-05	4.5E-07	7.3E-07	1.0E-06	1.7E-06
DIELDRIN	4.3E-08	1.0E-07	5.7E-04	1.3E-03	5.3E-06	1.2E-05	2.8E-08	6.6E-08
ENDRIN	1.0E-08	3.3E-08	1.2E-04	4.0E-04	1.1E-06	3.7E-06	6.0E-09	2.0E-08
FLUOROACETIC ACID	0.0E+00	0.0E+00	2.5E-03	2.9E-03	2.3E-05	2.7E-05	1.2E-07	1.4E-07
HEXACHLOROCYCLOPENTADIENE	1.0E-04	1.7E-04	3.4E-06	5.6E-06	3.2E-08	5.2E-08	5.2E-09	8.5E-09
ISODRIN	3.5E-05	1.3E-04	8.9E-04	3.3E-03	8.2E-06	3.1E-05	4.6E-08	1.7E-07
METHYLENE CHLORIDE	1.3E+00	1.7E+00	1.7E-05	2.2E-05	1.6E-07	2.1E-07	6.5E-05	8.5E-05
TETRACHLOROETHYLENE	1.1E-02	1.5E-02	2.1E-06	2.8E-06	2.0E-08	2.6E-08	5.7E-07	7.5E-07
ARSENIC	0.0E+00	0.0E+00	1.8E-03	4.1E-03	1.7E-05	3.8E-05	9.1E-08	2.0E-07
CADMIUM	0.0E+00	0.0E+00	1.9E-05	4.1E-05	1.8E-07	3.8E-07	9.4E-10	2.0E-09
COPPER	0.0E+00	0.0E+00	8.5E-04	3.0E-03	7.9E-06	2.8E-05	4.2E-08	1.5E-07
LEAD	0.0E+00	0.0E+00	7.9E-04	2.6E-03	7.3E-06	2.4E-05	3.9E-08	1.3E-07
MERCURY	0.0E+00	0.0E+00	3.2E-04	1.2E-03	3.0E-06	1.1E-05	1.6E-08	6.1E-08
ZINC	0.0E+00	0.0E+00	1.9E-03	5.6E-03	1.7E-05	5.2E-05	9.2E-08	2.8E-07

(1) - Total emission rate (volatile and wind erosion emission rates).

Mean represents the upper 95th percentile of the arithmetic mean, or reasonable maximum exposure (RME).

Max represents the maximum observed concentration for the contaminant.

The excavation emission rate ($E_{exc,i}$) for each COC is the product of the total suspended particulate loading factor, the excavation rate, and the soil concentration. Mathematically, this relationship may be expressed as shown:

$$E_{exc,i} = E_d * A * C_i \quad (5)$$

where

$E_{exc,i}$ = emission rate for component i (g/s);
E_d = total suspended particulate loading factor (kg/mg);
A = area of exposed soil (m^2);
C_i = concentration of contaminant i in soil (µg/g).

Appropriate conversion factors must be applied for consistency in the units.

Prior to calculating $E_{exc,i}$, the total suspended particulate loading rate must be computed as follows:

$$E_d = K * (0.0016) * \left(\frac{U}{2.2}\right)^{1.3} * \left(\frac{M}{2}\right)^{-1.4} \quad (6)$$

where

E_d = total suspended particulate loading factor (kg/mg);
K = particle size multiplier (0.48 dimensionless, for particles < 15 µm)
U = maximum 1-hour wind speed (m/s) 11 m/s was assumed;
M = soil moisture content (2.0 percent).

Excavation through the 10 ft section of potentially contaminated soil was assumed to take 1 hour. A particle size multiplier of 0.48 was employed together with a summer average soil moisture content for surface soils at RMA of 2 percent.

5.1.3.2 Atmospheric Dispersion

To calculate downwind air concentrations, the chemical-specific emission rates calculated above must be transported (i.e., dispersed through atmospheric transport) to specific receptor locations. For this RA, the closest receptor locations were selected for quantifying exposures and risks to on- and off-post receptors. The methodological approach and estimated downwind air concentrations are discussed below.

The Industrial Source Complex Short Term (ISCST) model (EPA, 1987a) was determined to be the most appropriate atmospheric dispersion model for this application. The ISCST model is a steady-state Gaussian plume model that can be used to calculate pollutant concentrations from a variety of sources.

The meteorological data were obtained from Stapleton Airport. Dispersion modeling typically uses a database large enough to include daily, seasonal, and annual variability (EPA, 1986). Five years (1983 through 1987) of sequential hourly data were used for this analysis. Meteorological parameters included wind speed, wind direction, temperature, cloud cover, ceiling heights, and mixing heights. The meteorological data were processed into a form compatible with the ISCST model. Wind speeds less than 1.0 m/s were set equal to 1.0 m/s. The wind directions were converted to flow vectors. Stability class (an indicator of the turbulence of the lower atmosphere) was derived from cloud cover, ceiling heights, wind speed, and time of day according to Turner's method (EPA, 1988b). Mixing heights (the height available for vertical mixing) were derived from upper air soundings at Stapleton Airport.

The LSB site was modeled using two different source areas to reflect the LSB IRA activities. The site was modeled as four industrial source areas (each 71 m on a side) centered as a group to reflect relocation of the lime sludge. Another set of calculations were performed using a 1-m^2 area source to reflect backhoe operations. The LSB site was modeled assuming a unit emission of 1.0 g/sec-m. This approach will produce an emission independent dispersion factor for each averaging period and each location. The dilution factor multiplied

by the actual emission rate of the pollutant of interest will yield a ground level concentration ($\mu g/m^3$).

Mean and maximum 8- and 24-hour concentrations resulting from airborne emissions from the LSB site were calculated at two locations: 3,500 m directly south of the site for off-post receptors and 225 m to the west/southwest of the site for on-post receptors. These two locations represent the off-post receptors in RSA 404 and the RMA Fire Department.

The LSB site, modeled with five years of Stapleton Airport meteorology according to the methods presented above, produces the highest 8- and 24-hour dilution factors for the two area sizes. The parameter values used to compute the on- and off-post downwind air concentrations are as follows:

On-Post (8-hour):

	At 240 degrees, 225 m
Site Area: 20,400 m^2	Dilution Factor: 6390000
Site-Source Area: 1 m^2	Dilution Factor: 775

Off-Post (24-hour):

	At 180 degrees, 3,500 m
Site Area: 20,400 m^2	Dilution Factor: 128500
Site-Source Area: 1 m^2	Dilution Factor: 6.49

Using the dilution factors and the estimated contaminant emission rates (Table 5-1), downwind contaminant concentrations in air at the fire station and the nearest RMA boundary (due south of LSB) were computed and are presented in Table 5-2.

Table 5-2 Estimated Exposure Point Concentration for On- and Off-Post Receptors (ug/m^3).

CHEMICAL	ON POST MEAN	ON POST MAX	OFF POST MEAN	OFF POST MAX
ALDRIN	6.0E-01	2.2E+00	1.2E-02	4.4E-02
BENZENE	2.8E+02	3.4E+02	5.6E+00	6.8E+00
CHLORDANE	5.7E-01	2.0E+00	1.1E-02	4.0E-02
CHLOROBENZENE	1.1E+01	1.6E+01	2.2E-01	3.3E-01
CHLOROFORM	9.0E+02	9.7E+02	1.8E+01	1.9E+01
P-CHLOROPHENYLMETHYL SULFIDE	3.6E-01	9.8E-01	7.3E-03	2.0E-02
P-CHLOROPHENYLMETHYL SULFONE	9.8E-01	1.8E+00	2.0E-02	3.7E-02
P-CHLOROPHENYLMETHYL SULFOXIDE	1.2E-01	3.4E-01	2.4E-03	6.7E-03
DDE	2.5E-02	9.0E-02	4.9E-04	1.8E-03
DDT	7.0E-03	2.5E-02	1.4E-04	5.0E-04
DIBROMOCHLOROPROPANE	2.7E-01	6.6E-01	5.4E-03	1.3E-02
DICYCLOPENTADIENE	6.6E+00	1.1E+01	1.3E-01	2.2E-01
DIELDRIN	1.8E-01	4.3E-01	3.7E-03	8.6E-03
ENDRIN	3.9E-02	1.3E-01	7.8E-04	2.6E-03
FLUOROACETIC ACID	8.0E-01	9.4E-01	1.6E-02	1.9E-02
HEXACHLOROCYCLOPENTADIENE	3.3E-02	5.5E-02	6.7E-04	1.1E-03
ISODRIN	3.0E-01	1.1E+00	5.9E-03	2.2E-02
METHYLENE CHLORIDE	4.2E+02	5.4E+02	8.4E+00	1.1E+01
TETRACHLOROETHYLENE	3.6E+00	4.8E+00	7.3E-02	9.6E-02
ARSENIC	6.0E-01	1.3E+00	1.2E-02	2.6E-02
CADMIUM	6.2E-03	1.3E-02	1.2E-04	2.6E-04
COPPER	2.7E-01	9.7E-01	5.4E-03	1.9E-02
LEAD	2.6E-01	8.3E-01	5.1E-03	1.6E-02
MERCURY	1.0E-01	4.0E-01	2.1E-03	7.9E-03
ZINC	6.0E-01	1.8E+00	1.2E-02	3.6E-02

Mean represents the upper 95th percentile of the arithmetic mean, or reasonable maximum exposure (RME).
Max represents the maximum observed concentration for the contaminant.

5.1.4 Estimated Contaminant Intake Rates

Contaminant intake rates were estimated for each COC based on volatile and fugitive dust emissions using the following equation and input parameters, as presented in EPA guidance (EPA, 1989a):

$$\text{Intake Rate (mg/kg-day)} = \frac{CA * IR * ET * EF * ED}{BW * AT} \quad (7)$$

where

CA_{vapor}	=	downwind contaminant concentration of contaminant i (mg/m^3)
$CA_{particulate}$	=	concentration in soil * particulate (PM_{10}) emission
IR	=	reference breathing rate (1.25 m^3/hr)
ET	=	exposure time (on-post: 8 hrs/day; off-post: 24 hrs/day)
EF	=	exposure frequency (60 days/yr)
ED	=	exposure duration (1 yr)
BW	=	reference body weight of an exposed individual (70 kg)
AT	=	averaging time (carcinogens: 25,550 days); (noncarcinogens: 365 days).

As shown above, contaminant intake rates were computed differently for the evaluation of noncarcinogenic and carcinogenic contaminants. For noncarcinogens, intakes are averaged over the duration of the exposure (1 year), while intakes of carcinogens are averaged over an entire lifetime (EPA, 1989a). For carcinogenic contaminants, current carcinogen RA guidance (EPA, 1986) assumes that a brief exposure to high doses of a carcinogen is equivalent to a low-dose averaged over a longer exposure period (i.e., a lifetime).

For this assessment, an 8-hour exposure time was considered appropriate for on-post, working populations, while a 24-hour exposure time was deemed appropriate for off-post residential populations. It was also conservatively estimated that all particulate matter is respirable (i.e., less than 10 microns in size) and retained in the pulmonary tract and all contaminants are completely (100 percent) absorbed. Estimated contaminant intake rates for carcinogens and noncarcinogens are presented in Table 5-3 for on- and off-post populations.

Table 5-3 Estimated Contaminant Intake Rates (mg/kg-day).

CHEMICAL	ONPOST		OFFPOST	
	MEAN	MAX	MEAN	MAX
ALDRIN*	2.0E-07	7.4E-07	1.2E-08	4.4E-08
BENZENE*	9.4E-05	1.1E-04	5.6E-06	6.8E-06
CHLORDANE*	1.9E-07	6.8E-07	1.1E-08	4.0E-08
CHLOROBENZENE	2.5E-04	3.8E-04	1.5E-05	2.3E-05
CHLOROFORM*	3.0E-04	3.3E-04	1.8E-05	2.0E-05
P-CHLOROPHENYLMETHYL SULFIDE	8.5E-06	2.3E-05	5.1E-07	1.4E-06
P-CHLOROPHENYLMETHYL SULFONE	2.3E-05	4.3E-05	1.4E-06	2.6E-06
P-CHLOROPHENYLMETHYL SULFOXIDE	2.8E-06	7.9E-06	1.7E-07	4.7E-07
DDE*	8.3E-09	3.0E-08	4.9E-10	1.8E-09
DDT*	2.3E-09	8.5E-09	1.4E-10	5.0E-10
DIBROMOCHLOROPROPANE*	9.0E-08	2.2E-07	5.4E-09	1.3E-08
DICYCLOPENTADIENE	1.5E-04	2.5E-04	9.3E-06	1.5E-05
DIELDRIN*	6.2E-08	1.4E-07	3.7E-09	8.6E-09
ENDRIN	9.3E-07	3.0E-06	5.5E-08	1.8E-07
FLUOROACETIC ACID	1.9E-05	2.2E-05	1.1E-06	1.3E-06
HEXACHLOROCYCLOPENTADIENE	7.8E-07	1.3E-06	4.7E-08	7.7E-08
ISODRIN	7.0E-06	2.6E-05	4.2E-07	1.6E-06
METHYLENE CHLORIDE*	1.4E-04	1.8E-04	8.4E-06	1.1E-05
TETRACHLOROETHYLENE*	1.2E-06	1.6E-06	7.4E-08	9.6E-08
ARSENIC*	2.0E-07	4.5E-07	1.2E-08	2.7E-08
CADMIUM*	2.1E-09	4.5E-09	1.2E-10	2.7E-10
COPPER	6.4E-06	2.3E-05	3.8E-07	1.4E-06
LEAD	6.0E-06	1.9E-05	3.6E-07	1.2E-06
MERCURY	2.5E-06	9.3E-06	1.5E-07	5.5E-07
ZINC	1.4E-05	4.2E-05	8.4E-07	2.5E-06

** indicates carcinogens*

Mean represents the upper 95th percentile of the arithmetic mean, or reasonable maximum exposure (RME).

Max represents the maximum observed concentration for the contaminant.

5.2 TOXICITY ASSESSMENT

The toxicity assessment is comprised of two components. The first, hazard identification, is intended to characterize the nature and extent of the health hazards associated with chemical exposures. The second, dose-response assessment, determines the relationship between the magnitude of exposure to a chemical and the increased likelihood of adverse health effects. Each of these components are discussed below.

5.2.1 Hazard Identification

Hazard identification is addressed through the development of a toxicological profile that discusses various health effects (e.g., acute and chronic toxicity, reproductive effects, and carcinogenicity) observed from chemical exposures in animal studies and, where available, human epidemiological studies. Toxicity profiles have been developed for each COC and are included in Appendix B. These profiles were compiled from current toxicological literature, and include the following information:

- Chemical and physical properties
- Summary of toxic effects to humans, laboratory animals, and wildlife
- Regulations and recommended guidelines for chemical exposures
- Dose-response assessment.

The information used in compiling the toxicity profiles was obtained from the following sources:

- Exposure Assessment Report for Rocky Mountain Arsenal (EA for RMA), Volumes II-III, Toxicity Assessment (EBASCO, 1990).
- Computerized literature searches of the following on-line databases: Medline (on-line biomedical bibliographic records); HSDB (Hazardous Substances Data Base); and IRIS (EPA's Integrated Risk Information System).

The EA for RMA contained toxicity profiles that were originally developed from information obtained from the EPA Office Waste Programs Enforcement (OWPE) and the U.S. Army Biomedical Research and Development Laboratory (USABRDL). This information was

supplemented by computerized literature searches of the Dialog and Chemical Information Systems databases and incorporated in the toxicity profiles in Appendix B.

5.2.2 Dose-Response Assessment

The goal of the dose-response assessment is to estimate an allowable intake rate for different routes of exposure, based on a rigorous review of the toxicological literature, which can be used as a benchmark for comparing the intake rates estimated in this assessment. Reference intake rates have been developed by EPA and are typically presented as reference doses (RfDs) for noncarcinogens and cancer potency factors (CPFs) for carcinogens. In compiling the dose-response estimates for COCs, two primary reference sources were utilized: EPA Integrated Risk Information System (IRIS) (EPA, 1990b), and EPA Health Effects Assessment Summary Tables (HEAST) (EPA, 1990a).

Quantitative dose-response estimates are generally estimated through an extrapolation from the high doses reported in studies of human epidemiology or laboratory animals, to the relatively low doses generally associated with environmental exposures. These values (i.e., RfDs or CPFs) are more generally available for the oral route of exposure, though inhalation values have been developed for some chemicals.

The general approach for determining dose-response values differs between carcinogenic and noncarcinogenic chemicals. A discussion of these differences as well as a general discussion on the basic methodology for determining dose-response values for each classification are presented in the subsections which follow.

5.2.2.1 Noncarcinogens

The general methodology used to derive reference intake rates (i.e., RfDs) for noncarcinogenic effects is based on the identification of no-observed-adverse-effect-levels (NOAELs) or lowest-observed-adverse-effect-levels (LOAELs) in appropriate human or animal studies with the incorporation of uncertainty (i.e., margin of safety factors). One or more of these safety factors are included in the derivation of the RfD based on considerations of the following: (1) the duration of the experimental exposure; (2) effects elicited (if any); (3) extrapolation

of the data to other species (i.e., to humans); and (4) sensitive subgroups (i.e., intraspecies variability). The general formula to compute an RfD is as follows:

$$\text{RfD (mg/kg-day)} = \frac{\text{NOAEL or LOAEL}}{\text{Uncertainty Factor}} \quad (8)$$

RfDs are generally developed for both short-term (i.e., subchronic) and long-term (i.e., chronic) exposures. Since exposure to contaminants will occur for a short period of time (approximately 60 days), only subchronic RfDs were used in this assessment.

Limited inhalation dose-response values were available for site COCs from the reference sources identified above. Specifically, appropriate RfD values were identified for only three of the 25 COCs addressed in this assessment. These are:

Chlorobenzene:	0.005 mg/kg-day
Dicyclopentadiene	6×10^{-4} mg/kg-day
Hexachlorocyclopentadiene:	2×10^{-4} mg/kg-day

The remaining 22 COCs did not have available inhalation RfD values, and therefore, hazard indices for these compounds could not be calculated. The 22 COCs without inhalation RfD values are:

- Aldrin
- Benzene
- Chlordane
- Chloroform
- p-Chlorophenylmethyl Sulfide
- p-Chlorophenylmethyl Sulfone
- p-Chlorophenylmethyl Sulfoxide
- Dichlorodiphenylethene (DDE)
- 1,1,1-Trichloro-2,2-bis (4-Chlorophenylethane) (DDT)
- Dibromochloropropane
- Dieldrin
- Endrin
- Fluoroacetic Acid
- Isodrin
- Arsenic
- Cadmium
- Copper
- Mercury
- Lead
- Zinc

5.2.2.2 Carcinogens

Carcinogenic chemicals are assumed to have a nonthreshold effect (i.e., no dose is considered to be entirely risk-free). Therefore, reference intake rates are based on EPA CPFs. Carcinogenic dose-response estimates are typically developed by extrapolating high doses used in animal studies or observed in human epidemiological studies to low doses encountered in environmental exposures using the conservative linearized multistage extrapolation model. The slope of this extrapolation curve is used to derive the CPF. The CPF computed by EPA is a plausible upper bound estimate of the probability of a response per unit intake of a chemical over a lifetime.

The cancer potency factors (inhalation) for COCs used in the LSB RA are summarized in Table 5-4.

5.3 RISK CHARACTERIZATION

The estimated contaminant intake rates computed in Section 5.1.5 are compared with the dose-response estimates to characterize potential risks to exposed individuals. Potential risks are computed and discussed separately for noncarcinogenic and carcinogenic contaminants.

5.3.1 Quantification of Noncarcinogenic Health Risks

Noncarcinogenic health risks for a given contaminant are characterized through the evaluation of a Hazard Quotient (HQ). The HQ may be expressed mathematically as

$$HQ = IR/RfD_i \quad (9)$$

where IR_i is the estimated daily intake rate for a given contaminant, and RfD_i is the corresponding reference dose.

Health risks resulting from simultaneous exposures to multiple contaminants are quantified through the determination of a Hazard Index (HI) as follows:

$$HI = HQ_1 + HQ_2 + ... + HQ_i \quad (10)$$

Table 5-4 Cancer Potency Factors for Contaminants

CHEMICAL	CANCER POTENCY FACTORS (mg/kg/day)
ALDRIN	17
BENZENE	0.029
CHLORDANE	1.3
CHLOROBENZENE	0
CHLOROFORM	0.0081
P-CHLOROPHENYLMETHYL SULFIDE	0
P-CHLOROPHENYLMETHYL SULFONE	0
P-CHLOROPHENYLMETHYL SULFOXIDE	0
DDE	0.34
DDT	0.34
DIBROMOCHLOROPROPANE	22
DICYCLOPENTADIENE	0
DIELDRIN	16
ENDRIN	0
FLUOROACETIC ACID	0
HEXACHLOROCYCLOPENTADIENE	0
ISODRIN	0
METHYLENE CHLORIDE	0.014
TETRACHLOROETHYLENE	0.0033
ARSENIC	15
CADMIUM	6.1
COPPER	0
LEAD	0
MERCURY	0
ZINC	0

Risks from multiple chemical exposures are assumed to be additive and do not address potential synergistic or antagonistic mechanisms. This approach assumes that simultaneous exposure to multiple contaminants at subthreshold levels (i.e., dose below which an adverse effect is not observed) could result in an adverse health effect. When the HI exceeds a value of one, there may be concern for potential adverse health effects. Chemicals which do result in an HI greater than one should be interpreted cautiously since (1) slopes of chemical-specific dose-response curves above the reference dose may vary greatly such that HQs may not be directly comparable among chemicals and (2) RfDs represent varying levels of confidence in the toxicity database and include different uncertainty or modifying factors. Therefore, though it is desirable not to exceed a HQ of 1, an exceedance for a chemical with a RfD incorporating high uncertainty/modification factors and designated as "low confidence" may be of lesser concern.

5.3.2 Quantification of Carcinogenic Health Risks

The carcinogenic risk of a contaminant is determined by multiplying the contaminant intake rate by the cancer potency factor, as shown below:

$$\text{Risk} = \text{CIR} \times \text{CPF} \qquad (11)$$

where CIR is the estimated contaminant daily intake rate in mg/kg-day (averaged over 70 years), and CPF is the cancer potency factor $(\text{mg/kg-day})^{-1}$. The carcinogenic risk represents the probability that an individual will develop cancer over a lifetime of exposure. Resultive cancer risks represent incremental or excess individual lifetime cancer risks (i.e., above the background incidence).

Carcinogenic health risks resulting from exposures to multiple carcinogens are considered additive such that the total carcinogenic risk may be computed as follows:

$$\text{Risk}_T = \Sigma_i \text{Risk}_i \qquad (12)$$

where $Risk_T$ is the total cancer risk (expressed as a unitless probability), and Risk, is the estimated upperbound cancer risk for a given contaminant. Since the cancer potency factors represent upper 95th percentile confidence limits of the probability of carcinogenic response, resulting risk estimates also represent upper bound determination. In other words, the true risk is not likely to exceed the risk estimate and is in fact likely to be less or perhaps even zero.

5.3.3 On-Post and Off-Post Evaluations

5.3.3.1 Noncarcinogenic Hazard Index

Table 5-5 presents mean and maximum noncarcinogenic HIs (based on mean and maximum soil concentrations) for both on-post (at the RMA Fire Department) and off-post (the nearest RMA boundary). As shown in the table, none of the computed HIs exceed one, the level indicating a concern for potential adverse health effects. However, it should be noted that since RfDs were available for only three contaminants (chlorobenzene, dicyclopentadiene, and hexachlorocyclopentadiene), the computed HIs may be underestimated.

5.3.3.2 Carcinogenic Risks

Table 5-6 presents mean and maximum predicted individual cancer risks to on- and off-post populations resulting from LSB IRA activities. These results indicate that given the conservative assumptions used in this assessment, both on- and off-post populations have a total cancer risk level in excess of the generally accepted point of departure of 1×10^{-6}, however they are well within the acceptance risk range of 10^{-4} to 10^{-7}. The COCs contributing to the risk level (in descending order) include aldrin, arsenic, benzene, chloroform, dibromochloropropane, methylene chloride, dieldrin, chlordane, cadmium, tetrachloroethylene, DDE, and DDT. The predominant pathway contributing to the risk level is fugitive dust emission.

5.4 UNCERTAINTY CONSIDERATIONS

The following section discusses the information and key assumptions used in the human health RA and how the uncertainties associated with their use affect the computed risk estimates. Although a rigorous quantitative evaluation of these uncertainties is beyond the scope of this assessment, it is important to consider, at least qualitatively, the effect that

Table 5-5 Noncarcinogenic Hazard Index for On- and Off-Post Populations.

CHEMICAL	ONPOST MEAN	ONPOST MAX	OFFPOST MEAN	OFFPOST MAX
ALDRIN	0.0	0.0	0.0	0.0
BENZENE	0.0	0.0	0.0	0.0
CHLORDANE	0.0	0.0	0.0	0.0
CHLOROBENZENE	5.0E-02	7.7E-02	3.0E-03	4.6E-03
CHLOROFORM	0.0	0.0	0.0	0.0
P-CHLOROPHENYLMETHYL SULFIDE	0.0	0.0	0.0	0.0
P-CHLOROPHENYLMETHYL SULFONE	0.0	0.0	0.0	0.0
P-CHLOROPHENYLMETHYL SULFOXIDE	0.0	0.0	0.0	0.0
DDE	0.0	0.0	0.0	0.0
DDT	0.0	0.0	0.0	0.0
DIBROMOCHLOROPROPANE	0.0	0.0	0.0	0.0
DICYCLOPENTADIENE	2.6E-01	4.2E-01	1.6E-02	2.5E-02
DIELDRIN	0.0	0.0	0.0	0.0
ENDRIN	0.0	0.0	0.0	0.0
FLUOROACETIC ACID	0.0	0.0	0.0	0.0
HEXACHLOROCYCLOPENTADIENE	3.9E-03	6.4E-03	2.4E-04	3.9E-04
ISODRIN	0.0	0.0	0.0	0.0
METHYLENE CHLORIDE	0.0	0.0	0.0	0.0
TETRACHLOROETHYLENE	0.0	0.0	0.0	0.0
ARSENIC	0.0	0.0	0.0	0.0
CADMIUM	0.0	0.0	0.0	0.0
COPPER	0.0	0.0	0.0	0.0
LEAD	0.0	0.0	0.0	0.0
MERCURY	0.0	0.0	0.0	0.0
ZINC	0.0	0.0	0.0	0.0
Total Hazard Index	3.1E-01	5.0E-01	1.9E-02	3.0E-02

Mean represents the upper 95th percentile of the arithmetic mean, or reasonable maximum exposure (RME).
Max represents the maximum observed concentration for the contaminant.

Table 5-6 Upperbound Carcinogenic Risk for On- and Off-Post Populations.

CHEMICAL	ONPOST		OFFPOST	
	MEAN	MAX	MEAN	MAX
ALDRIN	3.4E-06	1.3E-05	2.0E-07	7.5E-07
BENZENE	2.7E-06	3.3E-06	1.6E-07	2.0E-07
CHLORDANE	2.5E-07	8.8E-07	1.5E-08	5.3E-08
CHLOROBENZENE	0.0	0.0	0.0	0.0
CHLOROFORM	2.5E-06	2.6E-06	1.5E-07	1.6E-07
P-CHLOROPHENYLMETHYL SULFIDE	0.0	0.0	0.0	0.0
P-CHLOROPHENYLMETHYL SULFONE	0.0	0.0	0.0	0.0
P-CHLOROPHENYLMETHYL SULFOXIDE	0.0	0.0	0.0	0.0
DDE	2.8E-09	1.0E-08	1.7E-10	6.1E-10
DDT	7.9E-10	2.9E-09	4.7E-11	1.7E-10
DIBROMOCHLOROPROPANE	2.0E-06	4.8E-06	1.2E-07	2.9E-07
DICYCLOPENTADIENE	0.0	0.0	0.0	0.0
DIELDRIN	9.9E-07	2.3E-06	5.9E-08	1.4E-07
ENDRIN	0.0	0.0	0.0	0.0
FLUOROACETIC ACID	0.0	0.0	0.0	0.0
HEXACHLOROCYCLOPENTADIENE	0.0	0.0	0.0	0.0
ISODRIN	0.0	0.0	0.0	0.0
METHYLENE CHLORIDE	2.0E-06	2.6E-06	1.2E-07	1.5E-07
TETRACHLOROETHYLENE	4.0E-09	5.3E-09	2.4E-10	3.2E-10
ARSENIC	3.0E-06	6.7E-06	1.8E-07	4.0E-07
CADMIUM	1.3E-08	2.7E-08	7.5E-10	1.6E-09
COPPER	0.0	0.0	0.0	0.0
LEAD	0.0	0.0	0.0	0.0
MERCURY	0.0	0.0	0.0	0.0
ZINC	0.0	0.0	0.0	0.0
Total Risk	1.7E-05	3.6E-05	1.0E-06	2.1E-06

Mean represents the upper 95th percentile of the arithmetic mean, or reasonable maximum exposure (RME).
Max represents the maximum observed concentration for the contaminant.

various assumptions used throughout this analysis are likely to have on the final intake and risk values computed. Uncertainty considerations are discussed generally and more specifically in relation to site data used in each of the key steps in the RA: toxicity assessment, exposure assessment, and risk characterization in the sections that follow.

5.4.1 Toxicity Assessment

Toxicity data for chemicals found at waste sites are usually limited. Therefore, uncertainties are associated with the dose-response estimates employed in the analysis. The major sources of uncertainty that could over- or underestimate contaminant risks in the current analysis include:

- Extrapolation of dose-response data from effects observed at high doses to low concentrations typically encountered from human contact with the chemical in the environment may overestimate contaminant toxicity.
- The use of dose-response data from animal studies to predict effects in humans could over- or underestimate toxicity.
- As discussed in Section 5.2.2.1, the lack of inhalation RfD values for 22 of the 25 COCs at this site may result in an underestimate of actual noncarcinogen site risk estimates.

5.4.2 Exposure Assessment

Contaminant intakes computed in the exposure assessment also have a considerable amount of uncertainty inherent in their estimation. The major sources of uncertainty that could over- or underestimate the calculated intake rates include:

- The use of environmental data from the RI, while generally representative, may or may not sufficiently characterize soil contamination at specific discrete locations across the entire LSB.
- The use of the 95th percentile of the arithmetic mean and maximum measured contaminant concentrations may overestimate contaminant intake rates.
- The potential overestimation of risks from conservative assumptions in contaminant emission and transport models used to predict downwind concentrations.
- The use of standard assumptions, including body weights, exposure frequencies and durations, and media contact rates in computing intake rates may underestimate risks for some individuals, and may overestimate risks for others.

- Assuming 100 percent pulmonary retention of particulates and 100 percent absorption may overestimate actual exposures.

5.4.3 Risk Characterization

Risks estimated from contaminant intake rates may be under- or overestimated based on the uncertainties inherent in the methodology used in their computation. The major sources of uncertainty in the calculated cancer and noncancer risk estimates include:

- The assumption of dose additivity for noncarcinogenic compounds that do not induce the same type of effects, or that do not act by the same toxicological mechanism may underestimate risks.

- The assumption that risks for carcinogens can be summed equally without consideration of the weight-of-evidence categories (i.e., equal weight is given to known human carcinogens and suspected human carcinogens) could over- or underestimate risks.

- The lack of chemical-specific dose-response data for 22 of the 25 COCs, as discussed in Section 5.2.2.1, and the resultant inability to calculate risks for these compounds, may result in an underestimate of risks.

- The assumption that exposure to multiple chemicals is additive and not taking into account other mechanisms such as synergism or antagonism may result in an over- or underestimate of potential risks.

6.0 ECOLOGICAL RISK ASSESSMENT

The purpose of the Ecological Risk Assessment is to evaluate the probability of adverse biological and ecological effects related to potential site contamination during LSB IRA activities.

This section presents a qualitative evaluation of the potential short-term effects to biota (i.e., chemical-related and physical hazards) and likely fate of the COCs resulting from implementation of the LSB IRA. The following elements are addressed:

- Exposure assessment
- Toxicity assessment
- Risk characterization

6.1 EXPOSURE ASSESSMENT

The ecological exposure assessment for the LSB IRA qualitatively evaluates the biological resources that may be exposed to COCs. This includes a summary of the species potentially located in the LSB area, a discussion of contaminant transport mechanisms and a qualitative evaluation of biota exposures during IRA activities.

6.1.1 Potentially Exposed Biota Populations

A variety of terrestrial and aquatic ecosystems have been observed on RMA. Habitat alterations on RMA have led to an increase in diversity and abundance of several plant species. In some areas, a decrease in diversity has resulted from the persistence of early successional weedy species.

In the area of proposed LSB IRA activities, the vegetation is variable. Areas near the basins are either devoid of vegetation or dominated by annual grasses or weedy forbs, and have been severely disturbed by vehicular traffic, waste effluent drainage, or other nearby disposal activities. Some native grasses such as blue grama grass and sand dropseed have been observed in an area between the fire station and the basins.

The area defined by the LSB IRA does not directly impact any unique habitats, vulnerable vegetative communities such as wetlands, or unusual landscape features, such as floodplains.

The prairie, steppe, and savannah communities characteristic of the Great Plains region and existing on RMA support a variety of terrestrial fauna. The diversity of wildlife in this region is enhanced by the wide variety of habitats present. Abundant food, cover, and other habitat components improve reproductive success and support large populations. An inventory of RMA wildlife species and details on their distribution are found in the Biota RI (ESE, 1989), and the MKE report on wildlife resources of RMA (MKE, 1989a).

Seventeen species of raptors were observed on RMA between 1985 and 1989. Red-tailed hawks, and several species of owls are commonly observed wintering on RMA. In the winters of 1986 to 1987 and 1987 to 1988, several bald eagle (a Federally endangered species) subadults were observed feeding both in and near Section 36 (Basin A), within a few hundred yards of the LSB. Details of the bald eagle study on RMA are found in the 1986 to 1988 Bald Eagle Studies report (ESE, 1988).

Other top carnivores that have been observed or may be present near the LSB IRA implementation area include badgers, coyotes, red foxes, and long-tailed weasels. The major prey species for the top carnivores on RMA is the black-tailed prairie dog. The nearest active prairie dog colony to the IRA implementation area is approximately 800 to 1000 feet northwest of the basin. Pheasants and mourning doves are common upland game birds that are often seen in riparian, tall grass, and weedy vegetation types within or near the IRA implementation area.

Several species of rodents and small mammals are present in the vicinity of the IRA implementation areas. These small- and medium-sized mammals are for the most part primary consumers (herbivores) in the food chain, and are preyed upon by top carnivores.

Deer are commonly observed throughout South Plants south of the IRA area, and often are seen migrating north and along the Sanitary Sewer lines west of the LSB. Deer are primary

consumers (herbivores) in the food chain, and weakened or young deer may provide a food source for local carnivores.

Key *species of concern* during implementation of the LSB IRA include:

- Species listed as Federally threatened and endangered (T&E), or considered as a candidate species for listing by the U.S. Fish and Wildlife Service (USFWS)
- Species considered important components of RMA ecosystems (e.g., abundant prey for important species such as T&E species)
- Species that are economically important (e.g., game and pest species)

The terrestrial species that may potentially be affected by the LSB IRA have been selected from the Biota RI (ESE, 1989) and the Comprehensive Monitoring Program (CMP). These species are endemic to, or have potential access to, the IRA area and may be affected by the level of disturbance from the IRA.

The species selected for analyses are animal and invertebrate components of the terrestrial food web which have the greatest potential for exposure as a result of IRA activities. These species can be broken down into terrestrial-sedentary and terrestrial-mobile species, as noted in Table 6-1. These particular food webs were considered relevant to these IRA implementation activities because: (1) several avian predator nesting areas are within a reasonable distance of the IRA area; and (2) any excavated or otherwise disturbed materials may temporarily serve as a potentially contaminated food source (e.g., earthworms and insect larvae), otherwise unavailable to higher food web species. Vegetation cover in the area of IRA activities is sparse to nonexistent; therefore, ingestion of vegetation by herbivores is not considered a complete pathway, and is not addressed further in this RA.

Variations in food consumption during the year and at different life stages, as well as the timing of the IRA, may result in potential exposure to organisms other than those addressed above.

Table 6-1 Information Matrix for RMA Biota Potentially Impacted by Lime Settling Basins IRA

Targeted CMP-Biota Terrestrial Species	Taxonomic Group	Trophic Level	Game Species	Prey Item for Endangered Species	Widespread Distribution on RMA	Home Range Limited to RMA
Mobile Species						
Mule Deer/Adult	mammal	herbivore	X		X	
Mule Deer/Juvenile	mammal	herbivore	X		X	
Desert Cottontail	mammal	herbivore	X	X	X	X
Black-tailed Prairie Dog/Adult	mammal	herbivore		X		X
American Kestrel/ Egg	bird	-				X
American Kestrel/ Juvenile	bird	carnivore				X
Ring-necked Pheasant/Juvenile	bird	insectivore	X		X	X
Ring-necked Pheasant/Adult	bird	granivore	X		X	
Sedentary Species						
Deer Mouse/Adult	mammal	omnivore			X	X
Thirteen Lined Ground Squirrel	mammal	omnivore			X	X
Grasshopper	invertebrate	herbivore/ detritivore			X	X
Earthworm	invertebrate	detritivore				X

Targeted CMP-Biota Terrestrial Species	Taxonomic Group	Trophic Level	Game Species	Prey Item for Endangered Species	Widespread Distribution on RMA	Home Range Limited to RMA
Plants	plant	primary producer			X	X
• Cheatgrass	plant	primary producer				
• Kochia	plant	primary producer				
• Sunflower	plant	primary producer				
• Prickly lettuce	plant	primary producer				
• Morning glory	plant	primary producer				

6.1.2 Evaluation of Exposure Pathways

An evaluation of subchronic and potential acute exposures to biological receptors is presented in this section. A general discussion of the potential routes of exposure to ecological receptors during this IRA is also presented below.

Specific data regarding previous biota exposure to contaminants in the immediate vicinity of the LSB are available through the CMP (Stollar et al., 1990). Sampling during the CMP and Biota RI Program occurred immediately southwest of the LSB. Results of the 1988 CMP field program (Stollar et al., 1989) indicated that of five organochlorine pesticides (OCPs) analyzed, only dieldrin was detected. The compound was found in invertebrates, plants, and small mammals, at concentrations up to 8.1 µg/g. Arsenic was also detected in sunflowers at a concentration of 0.37 µg/g. Mercury was not detected in any biota species collected. Results of the 1989 CMP field program (Stollar et al., 1990) indicated the presence of dieldrin, endrin, and arsenic in invertebrates, plants, and small mammals. As in 1988 CMP results, dieldrin was the one analyte most often detected, at concentrations up to 6.1 µg/g (geometric mean = 0.8 µg/g). Arsenic was detected once in sunflowers, at a concentration of 2.3 µg/g, while endrin was detected once in grasshoppers (0.16 µg/g). The results of the 1988 and 1989 CMP biota field programs implemented adjacent to the LSB suggest that chronic exposures to biota are already occurring. Any exposures that would occur as a result of IRA activities would result in short-term or acute exposures. These pathways are described below.

In the LSB, potential sources of contamination include contaminated soils and contaminated sediments in areas of ponding. Contaminant sources in other environmental media are not applicable to this IRA. Transport from contaminant sources to biologic receptors is governed by several interrelated factors, including the chemical and physical properties of the source media and COCs; the physical processes that affect contaminant migration; and several biological factors including species, trophic level, age, food web, pattern of movement, reproductive frequency, and physiological behavior. The chemical and physical properties of the biota COCs are summarized in the chemical toxicity profiles, presented in Appendix B.

6.1.3 Exposure Routes and Physical Hazards

Exposure routes that may pose a threat to biota in the IRA area are soil or water ingestion, air or dust inhalation, dermal contact from temporarily excavated contaminated soils, and possibly ingestion of water contaminated with biota COCs. Since the area is sparsely vegetated, the exposed surface soils may result in an increased exposure of OCPs and metals to wildlife receptors through wind erosion. IRA activities may generate fugitive and volatile emissions, resulting in acute or subchronic impacts. These potential exposures would be expected to occur within the impact zone of the IRA activities. However, evidence of at least some exposures to nearby biota across several trophic levels suggests that chronic exposures would heavily outweigh the effects of a short duration, acute exposure.

Potential physical hazards to biota from the IRA would include excavation during slurry wall construction, compaction in areas of ingress and egress, consequential disturbance of small mammal habitat, and destruction of limited vegetative cover. However, given the brief duration of the planned IRA invasive activities, short-term impacts (i.e., acute effects) are not anticipated, provided that appropriate institutional controls (i.e., fencing) and mitigation actions are implemented.

6.3 TOXICITY ASSESSMENT

In accordance with the RAG (EPA, 1989a) and the EPA Region I Guidance for Public Health and Ecological Risk Assessments (EPA, 1989b), toxicity assessments were prepared for each of the COCs identified in this assessment.

The toxicity assessment is typically comprised of two elements. The first, hazard identification, is intended to characterize the nature and extent of biota health hazards associated with chemical exposures. The second, dose-response assessment, determines the relationship between the magnitude of exposure to a chemical and the occurrence of adverse health effects. Dose-response estimates have not been developed for biota as was done for humans, because such values have not been developed for subchronic types of exposures. The information comprising the hazard identification for each COC is discussed in the "Toxicity to Wildlife" section of each toxicity profile in Appendix B.

6.4 RISK CHARACTERIZATION

This section summarizes the results of the exposure and toxicity assessments to characterize the risks to biotic receptors from implementation of the IRA.

6.4.1 Qualitative Estimate of Risk

As indicated in the exposure assessment, allowable exposure criteria for biota and the lack of standardized data regarding biota intake rates from acute exposures are not conducive to making quantitative estimates of risk to biota. Therefore, a qualitative discussion of potential exposures to ecological receptors is presented here.

As indicated previously, exposures to biota from implementation of the LSB IRA are anticipated to be negligible given the small scale of the planned invasive soil activities (less than 5 acres), and the short-term duration of the LSB IRA. Primarily, the largest impacts that could occur to biota as a result of the IRA would be physical in nature (i.e., compaction, excavation, and temporary disturbance of habitat). Such disturbances can be mitigated. Therefore, the risk of adverse acute or subchronic ecological impacts as a direct result of the implementation of the LSB IRA is low.

6.4.2 Uncertainty Considerations

The characterization of risk to ecologic receptors includes several areas of uncertainty. The lack of standardized data for use in computing contaminant intake rates (i.e., quantitative dose-response estimates and potential carcinogenicity data) and a lack of quantitative risk assessment methodology led to a qualitative rather than quantitative risk assessment. Based on these inherent uncertainties, the potential risks to ecological receptors may be over- or underestimated.

6.4.3 Summary of Risks

In summary, although it is not possible to quantify the risks to ecological receptors as a result of LSB IRA activities, it is expected that exposures of COCs to biota will be low. Some of the LSB IRA activities could, however, result in increased contaminant transport, physical

disturbances to vegetation and wildlife habitats, and subsequent disruption of sensitive transient biota populations, such as avian predators and large mammals. Physical disturbances can be mitigated, and as such pose limited long-term impacts to biota populations near the LSB.

7.0 SOCIAL AND AESTHETIC IMPACTS

Social impacts is defined, for the purpose of this discussion, as activities which cause the interference of the enjoyment of an individual's environment. This may be thought of as an interference with "comfortable living" rather than loss, damage, or injury. The following sections discusses the impacts of IR activities on noise levels, unacceptable odor emissions, visual impairments and vehicular traffic.

7.1 NOISE IMPACTS

Noise is generally described as "unwanted sound" and, hence, it follows that people may wish to be protected from its undesirable effects. The Colorado Noise Abatement Statute requires local authorities to take action to restrict noise amounting to public nuisance. Local ordinances restrict construction noise levels (sound pressure levels or SPLs) to those specified for industrial zones [80 dB(A)[1] during the day and 75 dB(A) at night].

The noise generated from this interim action will be from the heavy equipment used for excavation and driving sheet pile. Heavy equipment (excavators, bulldozers, front-end loaders, etc.) generally have an SPL of 85 dB (Peterson, 1980). To determine the impact to receptors from the LSB IRA activities the following equation is used:

Sound Propagation Calculation (American Industrial Hygiene Association, 1982):

$$SPL = SWL - (20)\log(r) - 8\ dB \qquad (13)$$

where

SPL	=	sound pressure level (dB)
SWL	=	sound power level (dB)
r	=	distance from the noise source (m)

Solving for SWL with SPL = 85 dB gives you SWL = 93 dB

then setting SPL = 80 dB and solving for r gives r = 1.8 meters = 6 ft.

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[1] dB(A) is a sound level reading in decibels made on the A-weighted network of a sound level meter at slow response.

At approximately 6 ft from the source the SPL in hemispherical free field radiation will be 80 dB, which is the maximum noise level allowed by the State of Colorado in residential areas during the day during construction activities. The nearest population of concern is 2 miles or 10,560 feet from this interim action and, therefore, will not be affected.

7.2 ODOR IMPACTS

Activities associated with implementation of this IRA are not expected to produce unpleasant odors which would have a negative impact on the off-post receptors. This conclusion was based on the low concentrations of organic compounds and the distance of 2 miles to the nearest off-post receptors. It is anticipated the LSB IRA activities will result in odors no greater than those generated by similar activities in uncontaminated soils.

7.3 VISUAL IMPACTS

Awareness of air pollution often depends heavily upon visual perception. Many of the COCs cannot be seen, unless attached to dust. The dust generation from implementation of this IRA is expected to be controlled through dust suppression techniques and, therefore, is not expected to create any visual impacts to human populations off-post.

7.4 VEHICULAR IMPACTS

Vehicular traffic due to IRA activities will not impact local traffic routes because the small work force will not generate enough commuting trips over the work period to be noticeable, and all activity will be in a restricted area on the RMA. It is not anticipated that vehicular traffic generated as a result of implementation of this IRA will affect the level of service that nearby local roads provide.

8.0 CONCLUSIONS AND RECOMMENDATIONS

This RA has addressed potential health risks to on- and off-post human populations and to on-post ecological receptors resulting from implementation of the LSB IRA. The human health and ecological RAs were treated separately for the purposes of this report.

Results of the human health RA indicated an increase in the individual cancer risk to on-post receptors approximately 1.7×10^{-5} (i.e., approximately 2 chances in 100,000 of contracting cancer) and to off-post receptors of approximately 1.0×10^{-6} (i.e., approximately one chance in one million of contracting cancer). Much of this risk may be attributed to the conservative assumptions used in estimating downwind concentrations, predicting contaminant intakes, and the carcinogenic potency of each compound. However, it should be noted that although the risks exceed the point of departure of 10^{-6}, they are within the acceptable risk range of 10^{-4} to 10^{-7} (EPA, 1989a).

The following activities are recommended to reduce the potential risks to human health below the point of departure risk of 10^{-6}:

- Use of earth moving techniques to minimize dust generation
- Use of dust suppressant techniques during excavation and stockpiling activities.

The hazard index for noncarcinogens was less than one, indicating that noncarcinogenic human health effects are not likely from LSB IRA activities.

Results of the ecological RA indicated that physical hazards pose the greatest short-term risks to biota from implementation of the IRA. Short-term exposures to biota from COCs evident in the LSB pose less of a risk than chronic exposures to COCs over an extended period of time.

Potential impacts to biota can be minimized if the following activities are implemented:

- Limit ingress and egress of construction equipment to minimize habitat disturbance
- Limit the amount of ponding that could occur during construction, so as to limit exposure routes

- Limit the amount of ponding that could occur during construction, so as to limit exposure routes

- Place fencing or other institutional controls around the site to limit large animal and avian predator activities during implementation.

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APPENDIX A

APPENDIX A

APPENDIX A
PRELIMINARY RISK ASSESSMENT SCREEN

Background

Prior to the development of a detailed risk evaluation for the LSB, a preliminary screen was conducted to provide a worst-case assessment of the potential risks associated with LSB IRA activities. Had the screen indicated no exceedances of acceptable risk levels (10^{-6}) for both on- and off-post populations, no further risk evaluation would have been completed. However, since the screen did indicate exceedances of acceptable risk levels, an in-depth risk evaluation was performed, using a refined atmosphere dispersion model to better characterize actual exposures and potential risks to on- and off-post populations as a result of IRA activities. Details of this RA are provided in the attached report, whereas the results of the preliminary RA screen are discussed below.

Methodology

There are two major differences in the way the screen was conducted, compared to the RA. First, the screening RA for the LSB used the same input parameters as in the RA, except that focus was on maximum COC concentrations instead of representative concentrations were used. Second the screen estimated downwind contaminant concentrations using the EPA-SCREEN dispersion model (EPA, 1988) to predict worst-case exposure point concentrations.

This appendix describes the EPA-SCREEN dispersion model and presents the results of the screen.

Atmospheric Dispersion

Atmospheric dispersion of contaminants from the bulldozing of lime sludge was estimated using the Gaussian dispersion model EPA-SCREEN (EPA, 1988). EPA-SCREEN is a single source, short-term dispersion model that was used for calculating maximum 1-hour ground level concentrations at specified distances from the source. EPA-SCREEN may be used for point-source (stack) emissions, flare releases, and area emissions and accounts for specific meteorological conditions and local terrain variations. The model explicitly calculates the

effects of multiple reflections of the plume off the ground when calculating concentrations under limited mixing conditions. Dispersion coefficients as a function of downwind distance and stability parameters are also calculated. The model assumes that all particulate matter behaves as a gas and that deposition is negligible (Hanna, 1971).

The input parameters for the model are shown in Table A-1. Typical worst-case meteorological conditions [windspeed of 2.5 m/s and stable conditions (F)] were used in the dispersion calculation (Turner, 1970). The model was run in the "area" mode for an area of 5 acres, the total area of the Lime Settling Basins. The nearest on-post receptor was assumed to be the RMA Fire Station located at 225 m west-southwest of the LSB. The nearest off-post receptor was assumed to be located at the nearest downwind boundary of the RMA, approximately 3.5 kilometers south of the proposed activity. A receptor height of 1.5 m was used to reflect breathing zone concentrations.

The ambient concentrations of the contaminants were calculated separately for the gas phase and the particulate phase. To obtain downwind contaminant concentrations, the sum of the contaminant-specific emission rates for gas phase, fugitive, and excavation dust were multiplied by the dispersion coefficients. Contaminant intakes were computed using standard reference values. Noncarcinogenic and carcinogenic health risks were computed by comparing intakes to dose-response estimates. The following series of tables present input parameters (Table A-1), emission rates (Table A-2), downwind concentrations (Table A-3), contaminant intake rates (Table A-4) and noncarcinogenic and carcinogenic health risk estimates (Tables A-5 and A-6).

Table A-1. Parameter Values and Results of Dispersion Modeling.

Parameter	Units	Value
Emission Rate	g/s	1.0
Source Height	m	0.5
Area	m^2	20,400
Receptor Height	m	1.5
Distance to Receptor (on post/off post)	m	225/3,500
Rural/Urban Option		Rural
Wind Speed	m/s	2.5
Stability Class	dimensionless	F (stable)
Results		
Maximum 1 Hour Concentration (on post/off post)	$\mu g/m^3$ (based on emission rate of 1 g/s)	668/33.30

where:

g/s = grams/second
m = meters
m^2 = meters-squared
m/s = meters/second
$\mu g/m^3$ = micrograms/meters-cubed

Table A-2 Estimated Contaminant Emission Rates (g/sec)

CHEMICAL	VOLATILE EMISSION		WIND EROSION		EXCAVATION		EMISSION RATE TOTAL	
	MEAN	MAX	MEAN	MAX	MEAN	MAX	MEAN	MAX
ALDRIN	3.6E-05	1.3E-04	1.8E-03	6.7E-03	1.7E-05	6.2E-05	1.9E-03	6.9E-03
BENZENE	8.8E-01	1.1E+00	5.5E-05	6.7E-05	5.1E-07	6.2E-07	8.8E-01	1.1E+00
CHLORDANE	7.6E-06	2.7E-05	1.7E-03	6.2E-03	1.6E-05	5.8E-05	1.8E-03	6.3E-03
CHLOROBENZENE	3.4E-02	5.2E-02	1.5E-05	2.2E-05	1.3E-07	2.1E-07	3.4E-02	5.2E-02
CHLOROFORM	2.9E+00	3.1E+00	7.3E-05	7.8E-05	6.7E-07	7.2E-07	2.9E+00	3.1E+00
P-CHLOROPHENYLMETHYL SULFIDE	1.1E-03	2.9E-03	8.2E-05	2.2E-04	7.6E-07	2.1E-06	1.1E-03	3.1E-03
P-CHLOROPHENYLMETHYL SULFONE	3.0E-03	5.6E-03	1.4E-04	2.6E-04	1.3E-06	2.4E-06	3.1E-03	5.8E-03
P-CHLOROPHENYLMETHYL SULFOXIDE	1.8E-04	5.0E-04	2.0E-04	5.6E-04	1.9E-06	5.2E-06	3.8E-04	1.1E-03
DDE	1.8E-07	6.6E-07	7.6E-05	2.8E-04	7.1E-07	2.6E-06	7.7E-05	2.8E-04
DDT	4.7E-08	1.7E-07	2.1E-05	7.8E-05	2.0E-07	7.2E-07	2.2E-05	7.9E-05
DIBROMOCHLOROPROPANE	8.4E-04	2.1E-03	2.3E-06	5.6E-06	2.1E-08	5.2E-08	8.4E-04	2.1E-03
DICYCLOPENTADIENE	2.1E-02	3.4E-02	4.8E-05	7.9E-05	4.5E-07	7.3E-07	2.1E-02	3.4E-02
DIELDRIN	4.3E-08	1.0E-07	5.7E-04	1.3E-03	5.3E-06	1.2E-05	5.8E-04	1.3E-03
ENDRIN	1.0E-08	3.3E-08	1.2E-04	4.0E-04	1.1E-06	3.7E-06	1.2E-04	4.0E-04
FLUOROACETIC ACID	0.0E+00	0.0E+00	2.5E-03	2.9E-03	2.3E-05	2.7E-05	2.5E-03	2.9E-03
HEXACHLOROCYCLOPENTADIENE	1.0E-04	1.7E-04	3.4E-06	5.6E-06	3.2E-08	5.2E-08	1.1E-04	1.7E-04
ISODRIN	3.5E-05	1.3E-04	8.9E-04	3.3E-03	8.2E-06	3.1E-05	9.3E-04	3.5E-03
METHYLENE CHLORIDE	1.3E+00	1.7E+00	1.7E-05	2.2E-05	1.6E-07	2.1E-07	1.3E+00	1.7E+00
TETRACHLOROETHYLENE	1.1E-02	1.5E-02	2.1E-06	2.8E-06	2.0E-08	2.6E-08	1.1E-02	1.5E-02
ARSENIC	0.0E+00	0.0E+00	1.8E-03	4.1E-03	1.7E-05	3.8E-05	1.9E-03	4.2E-03
CADMIUM	0.0E+00	0.0E+00	1.9E-05	4.1E-05	1.8E-07	3.8E-07	1.9E-05	4.2E-05
COPPER	0.0E+00	0.0E+00	8.5E-04	3.0E-03	7.9E-06	2.8E-05	8.6E-04	3.0E-03
LEAD	0.0E+00	0.0E+00	7.9E-04	2.6E-03	7.3E-06	2.4E-05	8.0E-04	2.6E-03
MERCURY	0.0E+00	0.0E+00	3.2E-04	1.2E-03	3.0E-06	1.1E-05	3.3E-04	1.2E-03
ZINC	0.0E+00	0.0E+00	1.9E-03	5.6E-03	1.7E-05	5.2E-05	1.9E-03	5.6E-03

Mean represents the upper 95th percentile of the arithmetic mean, or reasonable maximum exposure (RME).
Max represents the maximum observed concentration for the contaminant.

Table A-3 Estimated Exposure Point Concentration for On- and Off-Post Receptors (ug/m^3).

CHEMICAL	ON POST MEAN	ON POST MAX	OFF POST MEAN	OFF POST MAX
ALDRIN	1.2E+00	4.6E+00	6.2E-02	2.3E-01
BENZENE	5.9E+02	7.1E+02	2.9E+01	3.5E+01
CHLORDANE	1.2E+00	4.2E+00	5.9E-02	2.1E-01
CHLOROBENZENE	2.3E+01	3.5E+01	1.1E+00	1.7E+00
CHLOROFORM	1.9E+03	2.0E+03	9.5E+01	1.0E+02
P-CHLOROPHENYLMETHYL SULFIDE	7.7E-01	2.1E+00	3.8E-02	1.0E-01
P-CHLOROPHENYLMETHYL SULFONE	2.1E+00	3.9E+00	1.0E-01	1.9E-01
P-CHLOROPHENYLMETHYL SULFOXIDE	2.5E-01	7.1E-01	1.3E-02	3.5E-02
DDE	5.2E-02	1.9E-01	2.6E-03	9.4E-03
DDT	1.5E-02	5.3E-02	7.2E-04	2.6E-03
DIBROMOCHLOROPROPANE	5.6E-01	1.4E+00	2.8E-02	6.9E-02
DICYCLOPENTADIENE	1.4E+01	2.3E+01	6.9E-01	1.1E+00
DIELDRIN	3.9E-01	9.0E-01	1.9E-02	4.5E-02
ENDRIN	8.2E-02	2.7E-01	4.1E-03	1.3E-02
FLUOROACETIC ACID	1.7E+00	2.0E+00	8.3E-02	9.7E-02
HEXACHLOROCYCLOPENTADIENE	7.0E-02	1.2E-01	3.5E-03	5.7E-03
ISODRIN	6.2E-01	2.3E+00	3.1E-02	1.2E-01
METHYLENE CHLORIDE	8.8E+02	1.2E+03	4.4E+01	5.7E+01
TETRACHLOROETHYLENE	7.7E+00	1.0E+01	3.8E-01	5.0E-01
ARSENIC	1.2E+00	2.8E+00	6.2E-02	1.4E-01
CADMIUM	1.3E-02	2.8E-02	6.4E-04	1.4E-03
COPPER	5.7E-01	2.0E+00	2.9E-02	1.0E-01
LEAD	5.4E-01	1.7E+00	2.7E-02	8.6E-02
MERCURY	2.2E-01	8.3E-01	1.1E-02	4.1E-02
ZINC	1.3E+00	3.8E+00	6.3E-02	1.9E-01

Mean represents the upper 95th percentile of the arithmetic mean, or reasonable maximum exposure (RME).

Max represents the maximum observed concentration for the contaminant.

Table A-4 Estimated Contaminant Intake Rates (mg/kg-day).

CHEMICAL	ONPOST		OFFPOST	
	MEAN	MAX	MEAN	MAX
ALDRIN*	4.2E-07	1.5E-06	6.2E-08	2.3E-07
BENZENE*	2.0E-04	2.4E-04	3.0E-05	3.6E-05
CHLORDANE*	4.0E-07	1.4E-06	5.9E-08	2.1E-07
CHLOROBENZENE	5.3E-04	8.1E-04	7.9E-05	1.2E-04
CHLOROFORM*	6.4E-04	6.9E-04	9.6E-05	1.0E-04
P-CHLOROPHENYLMETHYL SULFIDE	1.8E-05	4.9E-05	2.7E-06	7.3E-06
P-CHLOROPHENYLMETHYL SULFONE	4.9E-05	9.1E-05	7.3E-06	1.4E-05
P-CHLOROPHENYLMETHYL SULFOXIDE	6.0E-06	1.7E-05	8.9E-07	2.5E-06
DDE*	1.7E-08	6.3E-08	2.6E-09	9.4E-09
DDT*	4.9E-09	1.8E-08	7.3E-10	2.6E-09
DIBROMOCHLOROPROPANE*	1.9E-07	4.7E-07	2.8E-08	6.9E-08
DICYCLOPENTADIENE	3.3E-04	5.3E-04	4.9E-05	8.0E-05
DIELDRIN*	1.3E-07	3.0E-07	1.9E-08	4.5E-08
ENDRIN	1.9E-06	6.3E-06	2.9E-07	9.5E-07
FLUOROACETIC ACID	3.9E-05	4.6E-05	5.8E-06	6.9E-06
HEXACHLOROCYCLOPENTADIENE	1.7E-06	2.7E-06	2.5E-07	4.0E-07
ISODRIN	1.5E-05	5.5E-05	2.2E-06	8.2E-06
METHYLENE CHLORIDE*	2.9E-04	3.9E-04	4.4E-05	5.8E-05
TETRACHLOROETHYLENE*	2.6E-06	3.4E-06	3.8E-07	5.0E-07
ARSENIC*	4.2E-07	9.3E-07	6.2E-08	1.4E-07
CADMIUM*	4.3E-09	9.3E-09	6.4E-10	1.4E-09
COPPER	1.3E-05	4.8E-05	2.0E-06	7.1E-06
LEAD	1.3E-05	4.1E-05	1.9E-06	6.1E-06
MERCURY	5.1E-06	1.9E-05	7.6E-07	2.9E-06
ZINC	2.9E-05	8.8E-05	4.4E-06	1.3E-05

** indicates carcinogens*

Mean represents the upper 95th percentile of the arithmetic mean, or reasonable maximum exposure (RME).

Max represents the maximum observed concentration for the contaminant.

Table A-5 Noncarcinogenic Hazard Index for On- and Off-Post Populations.

CHEMICAL	ONPOST MEAN	ONPOST MAX	OFFPOST MEAN	OFFPOST MAX
ALDRIN	0.0	0.0	0.0	0.0
BENZENE	0.0	0.0	0.0	0.0
CHLORDANE	0.0	0.0	0.0	0.0
CHLOROBENZENE	1.1E-01	1.6E-01	1.6E-02	2.4E-02
CHLOROFORM	0.0	0.0	0.0	0.0
P-CHLOROPHENYLMETHYL SULFIDE	0.0	0.0	0.0	0.0
P-CHLOROPHENYLMETHYL SULFONE	0.0	0.0	0.0	0.0
P-CHLOROPHENYLMETHYL SULFOXIDE	0.0	0.0	0.0	0.0
DDE	0.0	0.0	0.0	0.0
DDT	0.0	0.0	0.0	0.0
DIBROMOCHLOROPROPANE	0.0	0.0	0.0	0.0
DICYCLOPENTADIENE	5.4E-01	8.9E-01	8.1E-02	1.3E-01
DIELDRIN	0.0	0.0	0.0	0.0
ENDRIN	0.0	0.0	0.0	0.0
FLUOROACETIC ACID	0.0	0.0	0.0	0.0
HEXACHLOROCYCLOPENTADIENE	8.3E-03	1.4E-02	1.2E-03	2.0E-03
ISODRIN	0.0	0.0	0.0	0.0
METHYLENE CHLORIDE	0.0	0.0	0.0	0.0
TETRACHLOROETHYLENE	0.0	0.0	0.0	0.0
ARSENIC	0.0	0.0	0.0	0.0
CADMIUM	0.0	0.0	0.0	0.0
COPPER	0.0	0.0	0.0	0.0
LEAD	0.0	0.0	0.0	0.0
MERCURY	0.0	0.0	0.0	0.0
ZINC	0.0	0.0	0.0	0.0
Total Hazard Index	6.6E-01	1.1E+00	9.8E-02	1.6E-01

Mean represents the upper 95th percentile of the arithmetic mean, or reasonable maximum exposure (RME).
Max represents the maximum observed concentration for the contaminant.

Table A-6 Upperbound Carcinogenic Risk for On- and Off-Post Populations.

CHEMICAL	ONPOST		OFFPOST	
	MEAN	MAX	MEAN	MAX
ALDRIN	7.1E-06	2.6E-05	1.1E-06	3.9E-06
BENZENE	5.7E-06	6.9E-06	8.6E-07	1.0E-06
CHLORDANE	5.1E-07	1.8E-06	7.7E-08	2.8E-07
CHLOROBENZENE	0.0	0.0	0.0	0.0
CHLOROFORM	5.2E-06	5.6E-06	7.7E-07	8.3E-07
P-CHLOROPHENYLMETHYL SULFIDE	0.0	0.0	0.0	0.0
P-CHLOROPHENYLMETHYL SULFONE	0.0	0.0	0.0	0.0
P-CHLOROPHENYLMETHYL SULFOXIDE	0.0	0.0	0.0	0.0
DDE	5.9E-09	2.1E-08	8.8E-10	3.2E-09
DDT	1.7E-09	6.0E-09	2.5E-10	9.0E-10
DIBROMOCHLOROPROPANE	4.2E-06	1.0E-05	6.2E-07	1.5E-06
DICYCLOPENTADIENE	0.0	0.0	0.0	0.0
DIELDRIN	2.1E-06	4.8E-06	3.1E-07	7.2E-07
ENDRIN	0.0	0.0	0.0	0.0
FLUOROACETIC ACID	0.0	0.0	0.0	0.0
HEXACHLOROCYCLOPENTADIENE	0.0	0.0	0.0	0.0
ISODRIN	0.0	0.0	0.0	0.0
METHYLENE CHLORIDE	4.1E-06	5.4E-06	6.2E-07	8.1E-07
TETRACHLOROETHYLENE	8.5E-09	1.1E-08	1.3E-09	1.7E-09
ARSENIC	6.3E-06	1.4E-05	9.4E-07	2.1E-06
CADMIUM	2.6E-08	5.7E-08	3.9E-09	8.5E-09
COPPER	0.0	0.0	0.0	0.0
LEAD	0.0	0.0	0.0	0.0
MERCURY	0.0	0.0	0.0	0.0
ZINC	0.0	0.0	0.0	0.0
Total Risk	3.5E-05	7.5E-05	5.3E-06	1.1E-05

Mean represents the upper 95th percentile of the arithmetic mean, or reasonable maximum exposure (RME).
Max represents the maximum observed concentration for the contaminant.

References

EPA (U.S. Environmental Protection Agency). 1988. Screening Procedures for Estimating the Air Quality Impact of Stationary Sources. Office of Air Quality Planning and Standards. Research Triangle Park, NC. EPA-4501-188-010.

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APPENDIX B
TOXICITY PROFILES

APPENDIX B
TOXICITY PROFILES

Toxicity Profiles were developed for each contaminant of concern evaluated for the Sanitary Sewers Interim Response Action. These profiles were developed to provide a brief overview of the toxicological properties and environmental behavior of the contaminants of concern to human and biota receptors at RMA. These profiles were not meant to be complete sources of toxicological or environmental information. These profiles were compiled from current toxicological literature, and include the following information:

- Chemical and physical properties
- Summary of toxic effects to humans, lab animals, and wildlife
- Regulations and standards
- Dose-response assessment

The information used in compiling the toxicity profiles was obtained from the following sources:

- Exposure Assessment for Rocky Mountain Arsenal, Volume I-II (Toxicity Assessment)
- USEPA Health Effects Assessment Summary Tables (July, 1990)
- Computerized literature searches of the following on-line databases:
    - IRIS (USEPA's Integrated Risk Information System - access date September, 1990)
    - Medline (on-line biomedical bibliographic records)
    - HSDB (Hazardous Substances Data Base)

The EA for RMA contained toxicity profiles that were originally developed from information obtained from the USEPA Office Waste Programs Enforcement (OWPE) and the U.S. Army Biomedical Research and Development Laboratory (USAMBRDL). This information was supplemented by computerized literature searches of the Dialog and Chemical Information Systems databases. These toxicity profiles were included in this risk assessment and updated using the sources listed above.

ACRONYM LIST FOR THE TOXICOLOGICAL PROFILES

ACGIH TLV:	American Conference of Governmental Industrial Hygienist Threshold Limit Values
CAS:	Chemical Abstract Number
EC_{50}	Median Effective Dose
IUPAC:	International Union of Pure and Applied Chemists
LC_{50}:	Median Lethal Concentration
LC_{LO}:	Lethal Concentration Low
LD_{50}:	Median Lethal Dose
mg/m^3	Milligram/cubic meter
ng/liter:	Nanogram/liter
NIOSH REL:	National Institute of Occupational Safety and Health Administration Recommended Exposure Limits
OSHA PEL:	Occupational Safety and Health Administration Permissible Exposure Limits
ppm:	Parts Per Million
STEL:	Short Term Exposure Limit
TWA:	Time Weighted average
µg/liter	Micrograms Per Liter

ALDRIN/DIELDRIN

SUMMARY

In the environment aldrin degrades to its persistent epoxide derivative dieldrin. Both pesticides have been shown to produce teratogenic and reproductive effects in animal studies. Aldrin and dieldrin have been shown to cause liver toxicity and central nervous system abnormalities in humans following chronic exposure. Both have been shown to be acutely toxic, with oral LD_{50} values ranging from 39-60 mg/kg in rats. Both pesticides are very toxic to aquatic organisms and have been associated with large-scale kills of terrestrial wildlife in treated areas.

CAS Number:	Aldrin:	309-00-2
	Dieldrin:	60-57-1
Chemical Formula:	Aldrin:	$C_{12}H_8Cl_6$
	Dieldrin:	$C_{12}H_8Cl_6O$
IUPAC Name:	Aldrin:	1,2,3,4,10,10-hexachloro-1,4,4a,5,8, 8a-hexahydro-1,4: 5,8-exo-dimethanonaphthalene
	Dieldrin:	1,2,3,4,10,10-hexachloro-6,7-expoxy-1,4,4a,5,6,7,8, 8a-octahydro-endo,exo-1,4:5,8-di methanonaphthalene

CHEMICAL AND PHYSICAL PROPERTIES

Molecular Weights:	Aldrin:	365
	Dieldrin:	381
Melting Point:	Aldrin:	104°C
	Dieldrin:	176°C
Solubility in Water:	Aldrin:	200 µg/liter at 25°C
	Dieldrin:	200 µg/liter at 25°C
Solubility in Organics:		Soluble in most organic solvents

Log Octanol/Water Partition Coefficient (K_{ow}):

Aldrin:
- 3.01 (Hansch, 1979)
- 5.66 (Geyer et al., 1984)
- 7.40 (Briggs, 1981)
- 5.66 (Kenaga, 1980, Table III)
- 5.30 (USEPA, 1986)

Dieldrin: 4.32 (Davies and Dobbs, 1984)
6.2 (Briggs, 1981)
3.69 (Rao and Davidson, 1983)
5.48 (Kenaga, 1980, Table III)
3.5 (USEPA, 1986)

Soil/Water Partition Coefficient (K_{oc}):

Aldrin: 76,000 (Versar, 1984)
28,200 (Briggs, 1981, Table III)
96,000 (USEPA, 1986)

Dieldrin: 3,300; 12,880 (Kadeg et al., 1986, literature values)
7,413 (Briggs, 1981, Table III, experimental)
35,600 (Kenaga, 1980, Table III, experimental)
1,700 (USEPA, 1986)

Bioconcentration Factor:

Aldrin: 1,555 (Davies and Dobbs, 1984, Eqn C, log k_{ow} = 5.66)
13,640 (Davies and Dobbs, 1984, Eqn C, log k_{ow} = 7.4)
1,500 (Lyman et al., 1982)
3,140 (Kenaga, 1980, Table 3, experimental)
10,800 (Kenaga, 1980, Table 3, experimental)
3,690 (Davies and Dobbs, 1984, Eqn B, log k_{ow} = 5.66)
40,345 (Davies and Dobbs, 1984, Eqn C, log k_{ow} = 7.4)
11,792 (Lyman et al., 1982, Eqn 5-2, log k_{ow} = 5.66)
247,742 (Lyman et al., 1982, Eqn 5-2, log k_{ow} = 7.4)
1,810 (Davies and Dobbs, 1984, Eqn C, log k_{ow} = 6.12)
6,940 (Davies and Dobbs, 1984, Eqn B, log k_{ow} = 6.12)
26,400 (Lyman et al., 1982, Eqn 5-2, log kow = 6.12)
4,571 (Hawker et al., 1986)

Dieldrin: 5,800; 4,420 (Kenga, 1980, Table 3, experimental)
1,489 (Davies and Dobbs, 1984, Eqn B, log k_{ow} = 5.0)
12,590 (Davies and Dobbs, 1984, Table 2, experimental)
292 (Davies an Dobbs, 1984, Eqn C, log k_{ow} = 4.32)
1,130 (Lyman et al., 1982, Eqn 5-2, log k_{ow} = 4.32)
30,339 (Lyman et al., 1982, Eqn 5-2, log k_{ow} = 6.2)
1,350.7 (Davies and Dobbs, 1984, Eqn a, S = 0.25)
480 (Davies and Dobbs, 1984, Eqn C, log k_{ow} = 5.0)
3,700 (Lyman et al., 1982, Eqn 5-2, log k_{ow} = 5.0)

Vapor Pressure: Aldrin: 2.31×10^{-5}mm Hg at 20°C
6×10^{-6}mm Hg (USEPA, 1986)

Dieldrin: 2.8×10^{-6}mm Hg at 20°C

Henry's Law Constant:

Aldrin:	2.4×10^{-5}atm-m^3/mole (calculated)
	1.6×10^{-5}atm-m^3/mole (USEPA, 1986)
Dieldrin:	1.4×10^{-5}atm-m^3/mole (calculated)
	4.58×10^{-7}atm-m^3/mole (USEPA, 1986)

TRANSPORT AND FATE

Photolysis occurs in aqueous solution or on plant surfaces, with conversion primarily to dieldrin, although a small fraction (generally less than 5 percent) is slowly converted to photodieldrin (Rosenblatt et al., 1975). Hydrolysis of dieldrin is also quite slow with a half-life in excess of 4 years (USEPA, 1979).

A range of experimental and estimated soil-water partition coefficients (K_{oc}) is reported above and indicates that substantial sorption of aldrin and dieldrin to soils/sediments and dissolved organic material will occur. Pavlou (1980) estimates that sorption of nonpolar hydrophobic pesticides is very high; therefore, little environmental mobility would be expected for these compounds.

In soil, aldrin is converted to its epoxide dieldrin, by oxidation. The conversion may be enhanced by microorganisms (Rosenblatt et al., 1975). The conversion appears to have a half-life on the order of 1 year. This degradation also occurs in vivo. The persistence of dieldrin in soil is variable but may range upwards of 7 years (Rosenblatt et al., 1975). Over 90 percent of applied dieldrin was still present in the top three inches of a loam soil after a period of 17 months (Rosenblatt et al., 1975). Microbial degradation does occur slowly with the main products being close derivatives (i.e., dihydroxydihydroaldrin, Rosenblatt et al., 1975).

Uptake of dieldrin in plants is variable. For example, potatoes grown in dieldrin treated soil had concentrations almost twice as high as soil levels (Telekar et al., 1983), while peeled beets had levels only one third the concentration in soil (Kohli et al., 1973). Concentrations in pasture crops appear to be less than the concentrations of the soil in which they were grown (Chawla et al., 1981).

A range of experimental and estimated bioconcentration factors (BCFs) for aldrin and dieldrin is also reported above. Bioconcentration factors for aldrin range from 1,500 to 247,000 indicating a high potential for biomagnification of residues up food chains. ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. Dieldrin is nonpolar and lipophilic, attracted to fats, plant waxes, and organic matter in sediments or soils (ESE, 1989), and it is expected that aldrin behaves similarly. Dieldrin does not leach readily, and neither mixing of soil nor adding organic matter appear to influence loss of residues through volatilization (ESE, 1989). It is expected that aldrin exhibits similar environmental behavior.

HEALTH EFFECTS

Both aldrin and dieldrin are probable carcinogens that have been shown to cause increases in a variety of tumors in rats at low but not at high doses. They also produce a higher incidence of liver tumors in mice. The reason for this reversed dose-response relationship is unclear. Neither appears to be mutagenic when tested in a number of systems. On the basis of the criteria proposed by the Carcinogen Assessment Group of the USEPA for evaluating the overall weight of evidence for carcinogenicity to humans, both aldrin and dieldrin are classified as Group B2 carcinogens (probable human carcinogens) (USEPA, 1989).

Aldrin and dieldrin have been shown to cause teratogenic and reproductive toxicity in animal test species. Reproductive effects include decreased fertility, increased fetal death, and effects on gestation. Teratogenic effects include cleft palate, webbed foot, and skeletal anomalies. Human chronic effects attributed to aldrin and dieldrin include liver toxicity and central nervous system abnormalities. Both chemicals are acutely toxic. The oral LD_{50} for aldrin in rats is 39-60 mg/kg (Merck, 1983). The oral LD_{50} for dieldrin in rats is 46 mg/kg (Merck, 1983). The dermal LD_{50} for both aldrin and dieldrin is approximately 100 mg/kg.

TOXICITY TO AQUATIC AND TERRESTRIAL WILDLIFE

Aquatic Organisms

Aldrin and dieldrin are both acutely toxic to freshwater species at low concentrations. Tests in fish showed that the two chemicals have similar toxicities, with LC_{50} values ranging from 1 to 53 µg/liter for different species. Final acute values (i.e., the concentration of material protecting 95 percent of the organisms; USEPA, 1980) for freshwater species were determined to be 2.5 µg/liter of dieldrin and 3.0 µg/liter for aldrin. Saltwater species were also quite sensitive to aldrin and dieldrin. The range of LC_{50} values was similar to that for freshwater species: 2 to 100 µg/liter for aldrin and 1 to 34 µg/liter for dieldrin. The saltwater Final Acute Values were 1.3 µg/liter for aldrin and 0.71 µg/liter for dieldrin.

Chronic studies have been conducted on the effects of dieldrin on freshwater and saltwater species. For freshwater organisms, chronic values as low as 0.2 µg/liter were obtained. The Final Acute Chronic Ratio was determined to be 8.5, and the calculated Freshwater Final Chronic Value was 0.29 µg/liter. Only one chronic study was done on saltwater species. Therefore, the saltwater Final Chronic Value of 0.084 µg/liter was determined by dividing the Final Acute Value by the Acute-Chronic Ratio. No chronic studies were identified for aldrin, but because its acute toxicity is comparable to that of dieldrin and because it is readily converted to dieldrin in animals and in the environment, it likely exhibits similar chronic toxicity.

Plants

Limited information was available in the literature reviewed regarding the phytotoxicity of aldrin. This information indicated that the uptake of organochlorine pesticides in plants following chronic exposure is variable, and that uptake is more likely in sandy soils than in soils with a high organic content.

Invertebrates

Earthworms were shown to concentrate aldrin-dieldrin residues up to 15 times the level found in field soil in long-term simulated ecosystem studies, and various insect species showed residue concentration factors of 11.9 to 58.4 times the soil level (ESE, 1989).

Birds

Aldrin has been associated with large scale bird kills in treated areas. Acute oral LD_{50} values for aldrin in bobwhite quail and pheasants were 6.59 and 16.8 mg/kg of body weight, respectively (Hudson et al., 1984). Aldrin causes liver toxicity and central nervous system disorders following chronic exposure, and has been shown to be acutely toxic to a variety of avian species.

Mammals

Both aldrin and dieldrin are acutely toxic with oral LD_{50} values ranging from 43 to 64 mg/kg in rats (ESE, 1989). Both pesticides have been associated with large scale mammal kills in treated areas and oral LD_{50} values for dieldrin in mule deer were 75 to 150 mg/kg body weight (Hudson et al., 1984). Both pesticides are associated with increased tumor induction in mice.

This pesticide has been shown to be carcinogenic in rats and mice, and is a teratogenic and reproductive toxicant.

REGULATIONS AND RECOMMENDED GUIDELINES

Ambient Water Quality Criteria (USEPA, 1986):

Aquatic Life (Freshwater)

Acute Toxicity:	Aldrin:	3.0 µg/liter
	Dieldrin:	2.5 µg/liter
Chronic Toxicity:	Aldrin:	No available data
	Dieldrin:	0.0019 µg/liter

Aquatic Life (Saltwater)

Acute Toxicity:	Aldrin:	1.3 µg/liter
	Dieldrin:	0.71 µg/liter

Chronic Toxicity: Aldrin: No available data
Dieldrin: 0.0019 µg/liter

Human Health

Due to the carcinogenicity of both aldrin and dieldrin the ambient water criterion is set at zero. However, estimates of the carcinogenic risks due to ingestion of both contaminated water and contaminated fish and shellfish are:

Risk	Aldrin Concentration	Dieldrin Concentration
10^{-5}	0.74 ng/liter	0.71 ng/liter
10^{-6}	0.074 ng/liter	0.071 ng/liter
10^{-7}	0.0074 ng/liter	0.0071 ng/liter

OSHA PEL 29 CFR 1910.1000 (air)*:

TWA = 0.25 mg/m^3 (skin designation)

NIOSH REL (air)*:

TWA = 0.15 mg/m^3

ACGIH TLV*:

TWA = 0.25 mg/m^3 (skin designation)

DOSE-RESPONSE ASSESSMENT

The human dose-response parameter estimates for carcinogens and noncarcinogens are computed differently by USEPA; therefore, these estimates are presented separately below.

Carcinogenic Effects

The Cancer Assessment Group (CAG) of the USEPA has derived an oral cancer potency estimate for aldrin of 1.7 x 10^1 mg/kg/day (USEPA, 1989). This estimate is based on the

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* Applies to both aldrin and dieldrin.

geometric mean of three potency slope factors determined for lower tumors in mice (Davis, 1965; Epstein, 1975; and NCI, 1978). The CAG has also derived an inhalation cancer potency estimate of 1.7×10^{1} mg/kg/day calculated from the oral data presented above (USEPA, 1989). The oral slope factor for dieldrin, a metabolite of aldrin, was determined to be essentially identical to that of aldrin (USEPA, 1989).

Aldrin Oral Cancer Potency Estimate:	1.7×10^{1} $(mg/kg/day)^{-1}$ (USEPA, 1989)
Aldrin Inhalation Cancer Potency Estimate:	1.7×10^{1} $(mg/kg/day)^{-1}$ (USEPA, 1989)

The inhalation cancer potency estimate was calculated from the oral cancer potency estimate (IRIS, 1989).

Noncarcinogenic Effects

The USEPA has computed chronic oral reference doses (RfD) of 3.0×10^{-5} (mg/kg/day) for aldrin/dieldrin (USEPA, 1989) based on 2-year chronic feeding study with rats, which identified a LOAEL (lowest-observed-adverse-effects-level) of 0.025 mg/kg/day (Fitzhugh et al., 1964). Higher doses produced liver lesions characteristic of chlorinated insecticide poisoning. An uncertainty factor of 1,000 was incorporated to account for uncertainties in extrapolating animal data to humans (10), to account uncertainties in the range of human sensitivities (10), and an additional uncertainty because the RfD is based on a LOAEL rather than a NOAEL (no-observed-adverse-effects-level [10]). An inhalation RfD is not currently available.

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ARSENIC

SUMMARY

Arsenic is a nonmetallic element that is present in the environment as a constituent of organic and inorganic compounds; it also occurs in a number of valence states. Arsenic is generally rather mobile in the natural environment, with the degree of mobility dependent on its chemical form and the properties of the surrounding media. Arsenic is a human carcinogen; it causes skin tumors when it is ingested and lung tumors when inhaled. Arsenic compounds are teratogenic and cause adverse reproductive effects in animals. Chronic exposure to arsenic is associated with polyneuropathy (disorders of the nervous system) and skin lesions. It is acutely toxic to some early life stages of aquatic organisms at levels as low as 40 µg/liter.

Arsenic can be found in the environment in any of four valence states (-3, 0, +3, and +5) depending on the pH, Eh, and other factors. It can exist as either inorganic or organic compounds and often will change forms as its moves through the various media. The chemical and physical properties depend on the state of the metalloid. Only the properties of elemental arsenic are presented below; properties of other arsenic compounds are often quite different.

CAS Number: 7440-38-2
Chemical Formula: As
IUPAC Name: Arsenic

CHEMICAL AND PHYSICAL PROPERTIES

Atomic Weight: 74.91
Boiling Point: 613°C
Melting Point: 817°C
Specific Gravity: 5.72 at 20°C
Solubility in Water: Insoluble; some salts are soluble

TRANSPORT AND FATE

In the natural environment arsenic has four different oxidation states; chemical speciation is important in determining arsenic's distribution and mobility. Interconversions of the +3 and +5 states as well as organic complexation do occur and can be mediated by microorganisms. Arsenic is generally quite mobile in the environment and is mainly transported by water (WHO, 1981). In oxygenated water, arsenic usually occurs as arsenate, but under reducing conditions (i.e., deep well waters) arsenite predominates. In the aquatic environment, volatilization is important when biological activity or highly reducing conditions produce arsine or methyl-arsenics. Sedimentation of arsenic in association with iron and aluminum does occur frequently (WHO, 1981).

In oxygenated soil, inorganic arsenic is prevalent in the pentavalent (+5) form. Under reducing conditions, the trivalent form predominates (WHO, 1981). Leaching of arsenates and arsenites occurs slowly due to binding with hydrous oxides of iron and aluminum. Biomethylation in soil does occur and may be associated with the release of methyl arsines into the air (WHO, 1981). Plant uptake of arsenic from treated soils can occur, however, accumulation is not excessive.

Freshwater residue data for arsenic (organic and inorganic) indicate that arsenic is not bioconcentrated to a high degree but that lower forms of aquatic life may accumulate higher residues than fish (USEPA, 1984a, 1986a).

HEALTH EFFECTS

Arsenic is an essential nutrient in humans and certain animal species. However, arsenic has been implicated in the production of skin cancer in humans. There is also extensive evidence that inhalation of arsenic compounds causes lung cancer in occupationally exposed workers. Arsenic compounds also cause noncancerous (possibly precancerous) skin changes in exposed individuals. However, arsenic has not definitively been found to be a carcinogen in animal studies (USEPA, 1984b). USEPA and the IARC have established that sufficient evidence exists to classify arsenic as a human carcinogen (USEPA, 1984b; IARC, 1980); it is therefore classified as a Group A carcinogen (i.e., human carcinogen) based upon evidence of human

carcinogenicity through inhalation and ingestion exposure. Arsenic compounds have been observed to cause chromosome damage in animals. Humans exposed to arsenic compounds have been reported to have an elevated incidence of chromosome aberrations.

Arsenic compounds have been reported to be teratogenic, fetotoxic, and embryotoxic in several animal species, and an increased incidence of multiple malformations among children born to women occupationally exposed to arsenic has been reported. Several cases of progressive polyneuropathy involving motor and sensory nerves and particularly affecting the extremities and myelinated long-axon neurons have been reported in individuals occupationally exposed to inorganic arsenic. Polyneuropathies have also been reported following the ingestion of arsenic-contaminated foods.

TOXICITY TO WILDLIFE AND DOMESTIC ANIMALS

Aquatic Life

Various inorganic forms of arsenic appear to have similar levels of toxicity. Inorganic arsenic appears to be more toxic than organic forms. Acute toxicity to adult freshwater animals occurs at levels of arsenic tiroxide as low as 812 µg/l and at levels as low as 40 µg/l in early stages of aquatic organisms. Acute toxicity to saltwater fish occurs at levels around 15 mg/l. arsenic toxicity does not appear to increase greatly with chronic exposure, and it does not seem that arsenic is bioconcentrated to a great degree.

Plants

Information was not found in the literature reviewed regarding the toxicity of arsenic to plants.

Invertebrates

Invertebrates were shown to be affected at much lower levels of arsenic as low as 508 µg/l.

Birds

Information was not found in the literature reviewed regarding the toxicity of arsenic to birds.

Mammals

Limited information was available in the literature reviewed regarding the toxicity of arsenic to mammals. Arsenic poisoning is an uncommon but not a rare toxic syndrome among domestic animals. Arsenic causes hyperemia (site-specific congestion) and edema (swelling) of the gastrointestinal tract, hemorrhage of the cardiac serosa surfaces and peritoneum, and pulmonary congestion and edema. It may also cause liver necrosis.

REGULATIONS AND RECOMMENDED GUIDELINES

Ambient Water Quality Criteria (USEPA, 1986a):

Aquatic Life (Freshwater)

Acute toxicity (As^{+3}): 360 µg/liter
Chronic toxicity (As^{+3}): 190 µg/liter

Aquatic Life (Saltwater)

Acute toxicity (As^{+3}): 69 µg/liter
Chronic toxicity (As^{+3}): 36 µg/liter

Human Health

Due to the carcinogenicity of arsenic the ambient water criterion is set at zero. However, estimates of the carcinogenic risks from the ingestion of contaminated water and contaminated aquatic organisms are:

Risk	Concentration
10^{-4}	222 ng/liter
10^{-5}	22 ng/liter
10^{-6}	2.2 ng/liter
10^{-7}	0.22 ng/liter

NIOSH REL:

15 MINUTE CEILING = 2 µg/m^3

OSHA PEL 29 CFR 1910.1018:

TWA = 10 µg/m^3

ACGIH TLV:

TWA = 200 $\mu g/m^3$

DOSE-RESPONSE ASSESSMENT

The human dose-response parameter estimates for carcinogens and noncarcinogens are computed differently by USEPA, therefore, these estimates are presented separately below.

Carcinogenic Effects

The Cancer Assessment Group (CAG) of USEPA has derived an oral cancer potency estimate for arsenic of 1.5 $(mg/kg/day)^{-1}$ (USEPA, 1986b). The CAG has also derived an inhalation cancer potency estimate of 15 (mg/kg/day)-1 (USEPA, 1990).

Oral Cancer Potency Estimate: 1.5 $(mg/kg/day)^{-01}$ (USEPA, 1988b)
Inhalation Cancer Potency Estimate: 15 $(mg/kg/day)^{-1}$ (USEPA, 1990)

Noncarcinogenic Effects

The USEPA has not currently derived an inhalation reference dose (RfD) for arsenic.

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BENZENE

SUMMARY

Benzene is an important industrial solvent and chemical intermediate. As benzene is volatile, atmospheric photooxidation is probably an important chemical fate process. Benzene is a known human carcinogen, causing leukemia in exposed individuals. It also adversely affects the hematopoietic system. Benzene has been shown to cause fetotoxicity and embryolethality in exposed experimental animals. Exposure to high concentrations of benzene in the air causes central nervous system depression and cardiovascular effects. Dermal exposure at high concentrations may cause dermatitis.

CAS Number: 71-43-2

IUPAC Name: Benzene

Chemical Formula: C_6H_6

CHEMICAL AND PHYSICAL PROPERTIES

Molecular Weight: 78.12

Boiling Point: 80.1°C

Melting Point: 5.56°C

Specific Gravity: 0.88 at 20°C

Solubility in Water: 1,780 mg/liter at 25°C
1,750 mg/liter at 25°C (USEPA, 1986a)

Solubility in Organics: Miscible with ethanol, ether, acetic acid, acetone, chloroform, carbon disulfide, and carbon tetrachloride

Log Octanol/Water Partition Coefficient (Kow): 2.01 (Valvani et al., 1980)
2.11 (Geyer et al., 1984)
2.12 (USEPA, 1986a)
2.13 (Moriguchi, 1975)

Soil/Water Partition Coefficient (Koc):

18-83 Sabljic (1984) Table II
83 Kenaga (1980) Table I

Bioconcentration Factor:

5.2	(USEPA, 1985b) (experimental)
24	(USEPA, 1980a) (experimental)
24	(Davies and Dobbs, 1984) Eqn B (log Kow = 2)
19.8	(Lyman et al., 1982) Eqn 5-2 (log Kow = 2.01)
23.6	(Lyman et al., 1982) Eqn 5-2 (log Kow = 2.11)
24.5	(Lyman et al., 1982) Eqn 5-2 (log Kow = 2.13)
18.5	(Davies and Dobbs, 1984) Eqn C (log Kow = 2.11)
9.3	(Davies and Dobbs, 1984) Eqn A (S = 1,700)
16.4	(Davies and Dobbs, 1984) Eqn C (log Kow = 2.13)
28.8	(Davies and Dobbs, 1984) Eqn B (log Kow = 2.13)

Vapor Pressure: 75 mm Hg at 20°C
95.2 mm Hg at 25°C (USEPA, 1985b)
100 mm Hg at 26°C (Perry and Chilton, 1973)

Vapor Density: 2.77

Henry's Law Constant: 0.006 atm-m^3/mole (calculated)
5.59×10^{-3} atm-m^3/mole (USEPA, 1986a)

Flash Point: -11.1°C

TRANSPORT AND FATE

Volatilization is the major transport process of benzene from surface waters to the ambient air and occurs readily (USEPA, 1979). Atmospheric breakdown of benzene is the most likely chemical fate process following its release to air. Although direct oxidation of benzene in environmental waters is unlikely, cloud chamber data indicate that it may be photooxidized rapidly in the atmosphere. The half-life of benzene in air is approximately 6 days (USEPA, 1986a). In surface waters, the estimated half-life ranges from 1-6 days (USEPA, 1986a).

A range of experimental and estimated Kocs is reported above and indicates that some sorption of benzene to soils/sediments and dissolved organic material will occur. Pavlou (1980) estimates that sorption of volatile organic compounds will range from low to moderate. The combined water solubility and low organic partitioning of benzene suggests that this compound will exhibit some degree of environmental mobility.

A range of estimated BCFs for benzene is also reported above. ASTM (1985) indicates that chemicals with BCFs less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors suggests that appreciable bioconcentration or biomagnification of benzene residues is not likely to occur.

HEALTH EFFECTS

Benzene is a recognized human carcinogen (IARC, 1982). Applying the criteria for weight of evidence proposed by the CAG of the USEPA (50 Federal Register 46948 Wed. Nov. 13, 1985), benzene is most appropriately designated as a Group A (human) carcinogen. Several epidemiological studies provide sufficient evidence of a causal relationship between benzene exposure and leukemia in humans. Benzene is a known inducer of aplastic anemia in humans, with a latent period of up to 10 years. Similar adverse effects on the blood-cell-producing systems occur in animals exposed to benzene. In both humans and animals, benzene exposure is associated with chromosomal damage, although it is not mutagenic in microorganisms. Benzene was fetotoxic and caused embryolethality in experimental animals.

Exposure to very high concentrations of benzene [about 20,000 ppm (66,000 mg/m^3) in air] can be fatal within minutes (IARC, 1982). The prominent signs are central nervous system depression and convulsions with death usually following as a consequence of cardiovascular collapse. Milder exposure can produce vertigo, drowsiness, headache, nausea, and eventually unconsciousness if exposure continues. Deaths from cardiac sensitization and cardiac arrhythmias have also been reported after exposure to unknown concentrations. Although most benzene hazards are associated with inhalation exposure, dermal absorption of liquid benzene may occur, and prolonged or repeated skin contact may produce blistering, erythema, and a dry, scaly dermatitis. The acute oral LD_{50} value of benzene in rats ranges from 3.4 g/kg (immature rats) to 5.6 g/kg (older adult rats, USEPA, 1980b).

TOXICITY TO WILDLIFE AND DOMESTIC ANIMALS

Aquatic Life

The EC_{50} (effective concentration) values for benzene in a variety of invertebrate and vertebrate freshwater aquatic species range from 5,300 µg/l to 386,000 µg/l (USEPA, 1980b). However, only values for the rainbow trout (5,300 µg/l) were obtained from a flow through test and were based on measured concentrations. Results based on unmeasured concentrations in static tests are likely to underestimate toxicity for relatively volatile compounds like benzene. A chronic toxicity test with Daphina Magna was incomplete; however, no adverse effects were observed at test concentrations as high as 98,000 µg/l. For saltwater species, acute values for one fish and five invertebrate species range from 10,900 µg/l to 924,000 µg/l (USEPA, 1980b).

Plants

Freshwater and saltwater plant species that have been studied exhibit toxic effects at benzene concentrations ranging from 20,000 µg/l to 525,000 µg/l (USEPA, 1980b).

Invertebrates

Information, other than was given in the Aquatic Life section was not found in the literature reviewed regarding toxicity of benzene to invertebrates.

Birds

Information was not found in the literature reviewed regarding toxicity of benzene to birds.

Mammals

Information was not found in the literature reviewed regarding toxicity of benzene to mammals.

REGULATIONS AND RECOMMENDED GUIDELINES

Ambient Water Quality Criteria (USEPA, 1986b):

The available data are not adequate for establishing criteria. However, USEPA does report the lowest concentrations of benzene known to cause toxic effects in aquatic organisms.

Aquatic Life (Freshwater)

Acute toxicity: 5,300 ug/liter
Chronic toxicity: No available data

Aquatic Life (Saltwater)

Acute toxicity: 5,100 ug/liter
Chronic toxicity: No available data

Human Health

Due to the carcinogenicity of benzene, the ambient water criterion is set at zero. However, estimates of the carcinogenic risks associated with lifetime exposure from ingestion of contaminated water and contaminated aquatic organisms are:

Risk	Concentration
10^{-4}	66.6 ng/liter
10^{-5}	6.6 ng/liter
10^{-6}	0.66 ng/liter
10^{-7}	0.066 ng/liter

National Primary Drinking Water Standard: 0.005 mg/l (40 CFR Part 141)

OSHA PEL 29 CFR 1910.1028

TWA = 1 ppm

STEL = 5 ppm

ACGIH TLV:

TWA = 10 ppm

DOSE RESPONSE ASSESSMENT

The human dose-response parameter estimates for carcinogens and noncarcinogens are computed differently by USEPA; therefore, these estimates are presented separately below.

Carcinogenic Effects

The Cancer Assessment Group (CAG) of USEPA has derived an oral cancer potency estimate for benzene of 2.9×10^{-2} $(mg/kg/day)^{-1}$ (USEPA, 1990). The CAG has also derived an inhalation cancer potency estimate of 2.9×10^{-2} $(mg/kg/day)^{-1}$ (USEPA, 1990).

Oral Cancer Potency Estimate: 2.9×10^{-2} $(mg/kg/day)^{-1}$ (USEPA, 1990)
Inhalation Cancer Potency Estimate: 2.9×10^{-2} $(mg/kg/day)^{-1}$ (USEPA, 1990)

Noncarcinogenic Effects

The USEPA has not currently derived a reference dose (RfD) for benzene.

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CADMIUM

SUMMARY

Cadmium is a metal that is present in a variety of chemical forms in the environment. Cadmium has a valence of +2 and has properties similar to those of zinc. Cadmium forms both organic and inorganic compounds. Cadmium sulfate is the most common salt. Some forms are insoluble in water, but cadmium is relatively mobile in the aquatic environment. Cadmium is carcinogenic in animals exposed by inhalation and may also be in humans. It is uncertain whether it is carcinogenic in animals or humans following oral exposure. Cadmium is a known animal teratogen and reproductive toxin. It has chronic effects on the kidney, and background levels of human exposure are thought to provide only a relatively small margin of safety for these effects.

CAS Number: 7440-43-9
Chemical Formula: Cd
IUPAC Name: Cadmium

CHEMICAL AND PHYSICAL PROPERTIES

Atomic Weight: 112.41
Boiling Point: 765°C (Fleischer et al., 1974)
Melting Point: 321°C (Fleischer et al., 1974)
Specific Gravity: 8.642
Solubility in Water: Salts are water soluble; metal is insoluble
Solubility in Organics: Variable, based on compound
Vapor Pressure: 1 mm Hg at 394°C

TRANSPORT AND FATE

Cadmium is relatively mobile in the aquatic environment compared to other heavy metals (USEPA, 1979). It is removed from aqueous media by complexing with organic materials. The solubility of cadmium in equilibrium with mineralized soil is primarily controlled by carbonate and to a lesser extent by hydroxyl ion (USEPA, 1979). Minimum levels of

cadmium solubility occur at pH levels of nine or above. It appears that cadmium moves slowly through soil, but only limited information on soil mobility is available. Organic constituents of soil apparently can adsorb cadmium from aqueous systems.

In general, cadmium is readily concentrated into vegetable plant matter. However, concentration effects may be confounded by the onset of phytotoxic effects. Uptake in aquatic weeds is variable, with bioconcentration factors ranging between 600-1200 (USEPA, 1980). Bioconcentration factors for cadmium in freshwater aquatic life range from 164 to 4,190 for invertebrates and from 3 to 2,213 for fishes (USEPA, 1986). ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the reported concentration factors suggests that appreciable bioconcentration or biomagnification of cadmium may occur.

HEALTH EFFECTS

There is suggestive evidence linking cadmium with cancer of the prostate in humans (USEPA, 1980). In animal studies, exposure to cadmium by inhalation caused lung tumors in rats, and exposure by injection produced injection-site sarcomas and/or Leydig-cell tumors (Takenaka et al., 1983; USEPA, 1981). An increased incidence of tumors has not been seen in animals exposed to cadmium orally, but four of the five available studies were inadequate by current standards (Clement Associates, Inc., 1983). Cadmium has been classified in USEPA's Group B1 according to USEPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon evidence of carcinogenicity in humans through inhalation exposure.

The evidence from a large number of studies on the mutagenicity of cadmium is equivocal, and it has been hypothesized that cadmium is not directly mutagenic but may impede repair mechanisms (Clement Associates, Inc., 1983). Cadmium is a known animal teratogen and reproductive toxin. It has been shown to cause renal dysfunction in both humans and animals. Other toxic effects attributed to cadmium include immunosuppression (in animals), anemia (in humans), pulmonary disease (in humans), pulmonary edema and pneumonitis, possible effects

on the endocrine system, defects in sensory function, and bone damage (Casarett and Doull's Toxicology, 1980). The oral LD_{50} in the rat is 225 mg/kg (NIOSH, 1982).

TOXICITY TO WILDLIFE AND DOMESTIC ANIMALS

Aquatic Life

Laboratory experiments suggest that cadmium may have adverse effects on reproduction in fish at levels present in lightly to moderately polluted waters. The acute LC_{50} for freshwater fish and invertebrates generally ranged from 100 to 1,000 µg/l. Saltwater species were in general ten-fold more tolerant to the acute effects of cadmium; however, salmonids appear to be much more sensitive to cadmium than other organisms (USEPA, 1980). Chronic tests have shown that cadmium toxicity may be cumulative. The acute-chronic ratios for cadmium range from 66 to 431. Bioconcentration factors were generally less than 1,000, but as high as 10,000 for some freshwater fish species.

Plants

Information was not found in the literature reviewed regarding toxicity of cadmium to plants.

Invertebrates

Information was not found in the literature reviewed regarding toxicity of cadmium to invertebrates.

Birds

Cadmium chloride in the diets of quail, pheasants, or ducks resulted in LC_{50} values of 2,584, 767, and 5,000 ppm, respectively (Hillet al., 1975).

Mammals

Information was not found in the literature reviewed regarding toxicity of cadmium to mammals.

REGULATIONS AND RECOMMENDED GUIDELINES

Ambient Water Quality Criteria (USEPA, 1986):

Aquatic Life (Freshwater)

Acute toxicity: $e^{[1.30\ (\ln(\text{hardness})) - 3.92]}$ ug/liter
At hardness of 50, 100, and 200 mg/liter CaCO3, Criteria = 1.8, 3.9, and 8.6 ug/liter.

Chronic toxicity: $e^{[0.87\ (\ln(\text{hardness})) - 4.38]}$ ug/liter
At hardness of 50, 100, and 200 mg/liter CaCO3, Criteria = 0.66, 1.1, and 2.0 ug/liter.

Aquatic Life (Saltwater)

Acute toxicity: 43 ug/liter
Chronic toxicity: 9.3 ug/liter

Human Health

Criterion: 10 ug/liter [CAG Potency Slope for Inhalation Exposure (USEPA, 1990a): 6.1 $(mg/kg/day)^{-1}$]

National Primary Drinking Water Standard:

10 ug/liter (Proposed RMCL; 50 FR 46966 Wednesday, November 13, 1985 = 5 ug/liter)

NIOSH REL:

As low as achievable

OSHA PEL 29 CFR 1910.1000

TWA = 0.2 mg/m^3

Ceiling = 0.6 mg/m^3

ACGIH TLV

TWA = 0.05 mg/m^3

DOSE-RESPONSE ASSESSMENT

The human dose-response parameter estimates for carcinogens and noncarcinogens are computed differently by USEPA; therefore, these estimates are presented separately below.

Carcinogenic Effects

The Cancer Assessment Group (CAG) of USEPA has derived an inhalation cancer potency estimate for cadmium of 6.1 $(mg/kg/day)^{-1}$ (USEPA, 1990a). This estimate was based on occupational exposure data.

Inhalation Cancer Potency Estimate: 6.1 $(mg/kg/day)^{-1}$ (USEPA, 1990a).

Noncarcinogenic Effects

The oral intake reference dose (RfD) for cadmium is based on the RfD for water reported in the Health Effects Assessment Summary Table (HEAST) (USEPA, 1990b). The value reported by USEPA is for two media, food (1×10^{-3} mg/kg/day) and water (5×10^{-4} mg/kg/day). Details of the underlying study were not available.

Oral (food) RfD = 1×10^{-3} mg/kg/day
Oral (water) RfD = 5×10^{-4} mg/kg/day

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CHLORDANE

SUMMARY

Chlordane is an organochlorine pesticide that was formerly used on field crops and is presently used to control structural pests in homes. Technical chlordane is a complex mixture that includes two isomers of chlordane, heptachlor, and two isomers of nonachlor. It is very persistent in the environment and is readily bioaccumulated in fish and other aquatic organisms. Chlordane causes liver tumors in mice, and the results of a mutagenicity assay were positive. It causes adverse reproductive effects in mice, and chronic exposure causes liver changes and adversely affects the central nervous system. Chlordane is very toxic to aquatic organisms.

Technical Chlordane is a complex mixture, however, the major components are cis-Chlordane and trans-Chlordane. The technical product also contains a variety of other chlorinated hydrocarbons, including heptachlor. It is a viscous amber-colored liquid. Much of the available literature does not distinguish between the Chlordane isomers and appears to discuss mixtures of these compounds.

CAS Number: Chlordane (mixture): 57-74-9
cis-Chlordane: 5103-74-2
trans-Chlordane: 5103-71-9

Chemical Formula: $C_{10}H_6C_{18}$

IUPAC Name: 1,2,4,5,6,7,8,8-Octachloro-2,3,3a,4,7,7a-hexahydro-4, 7-methanoindene

Important Synonyms and Trade Names: cis-chlordane; alpha-chlordane trans-chlordane; gamma-chlordane

CHEMICAL AND PHYSICAL PROPERTIES

Molecular Weight: 409.3

Boiling Point: 175°C at 2 mm Hg

Melting Point: cis-Chlordane: 107-109°C, trans-Chlordane: 103-105°C

Specific Gravity: 1.59-1.635 at 16°C (technical Chlordane)

Solubility in Water: 0.056 to 1.85 mg/liter at 25°C

Solubility in Organics: Miscible in aliphatic and aromatic solvents (technical Chlordane)

Log Octanol/Water Partition Coefficient (Kow):

2.78; 3.32; 5.48 (Kadeg et al., 1986) literature values

Soil-Water Partition Coefficient (Koc):

422; 53,570	(Lyman and Loreti, 1987) (log Kow = 2.78; 5.48)
21,300	(Kenaga, 1980) Table III
624; 53,850	(Kadeg et al., 1986) (log Kow = 2.78; 5.48)
141,200	(Kadeg et al., 1986) (literature value)
140,000	(USEPA, 1986)
775; 22,810	(Lyman et al., 1982) Eqn 4-8 (log Kow = 2.78; 5.48)

Bioconcentration Factor: 14,000 (USEPA, 1968)

Vapor Pressure: 1×10^{-5} mm Hg at 20°C (USEPA, 1986)

Flash Point: Minimum 81°C (technical Chlordane)

Henry's Law Constant: 9.6×10^{-5} atm-m^3/mole (calculated)
9.63×10^{-6} atm-m^3/mole (USEPA, 1986)
4.05×10^{-4} Dimensionless

TRANSPORT AND FATE

Chlordane is very persistent in the environment, resisting chemical and biological degradation into less harmful substances. Chlordane is virtually insoluble in water. Chlordane present in clear water may be somewhat volatile, and this may be an important loss process. Less loss of Chlordane from aquatic systems will occur when organics are present due to adsorption processes. Therefore, residue concentrations in sediment are often much higher than in water.

Chlordane binds tightly to soil particles and persists for years in soils after surface application. A range of experimental and estimated Kocs is reported above and indicates that sorption of Chlordane to soils/sediments and dissolved organic material will occur. Pavlou (1980) estimates that sorption of organochlorine pesticides is very high; therefore, little environmental mobility would be expected for this compound.

Chlordane applied as an emulsifiable concentrate is more readily volatilized than when applied as a granular formulation. Certain food and feed crops can accumulate residues by absorption

from the soil. Chlordane has been found to accumulate in the peels of root vegetables studied (Rosenblatt et al., 1975). The persistence (half-life) of Chlordane in soil ranges from 2 to greater than 13 years (Rosenblatt et al., 1975). Atmospheric transport of vapors and contaminated dust particles from soil application sites can occur.

Chlordane exhibits strong tendencies for bioaccumulation in some aquatic and terrestrial organisms. It can concentrate at levels thousands of times greater than the surrounding water medium in a variety of aquatic organisms, including bacteria, algae, daphnids, and fish (USEPA, 1980). A range of estimated BCFs for Chlordane is presented above. ASTM (1985) indicates that chemicals with BCFs less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors suggests that bioconcentration or biomagnification of chlordane residues will occur.

HEALTH EFFECTS

Mice fed diets containing analytical-grade chlordane for 80 weeks exhibited a highly significant dose-dependant incidence of liver tumors (males and females). Positive results have also been reported in carcinogenicity tests with female rats (50 Federal Register 46988, Wed., Nov. 13, 1985b). Chlordane has been classified in USEPA's Group B2, according to USEPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon the positive results of these studies (50 Federal Register 46988, Wed., Nov. 13, 1985b). Chlordane has induced mutagenic effects in at least one test system. Negative results were obtained in chromosome aberration tests utilizing Chinese hamster ovary cells (NTP, 1985); however, positive evidence of sister chromatid exchange was obtained in the same test medium.

Reproductive effects, including developmental defects and neonatal metabolic and biochemical disorders, are observed in the offspring of mice exposed to Chlordane. Tests with laboratory animals, primarily rodents, have demonstrated acute and chronic toxic effects. Mixtures of the two isomers appear to exhibit similar toxicities to that of single isomers. Chronic exposure to chlordane causes liver changes and induces or suppresses a variety of enzyme systems. In addition, chlordane may act as a cumulative neurotoxin.

Acute effects include anorexia, weight loss, tremors, convulsions, and death. The oral LD_{50} in the rat is 283 mg/kg. Oxychlordane, an epoxide metabolite formed from either Chlordane isomer, is more acutely toxic than Chlordane. The oral LD_{50} of oxychlordane administered to rats in corn oil is 19 mg/kg, and 43 mg/kg when administered in an aqueous suspension.

Clinical symptoms of acute oral or dermal exposure to Chlordane in humans include vomiting, seizures, electroencephalographic dysrhythmia, convulsions, and possible death. Oxychlordane has been found in a high percentage of sampled human adipose tissues and also in milk samples.

TOXICITY TO WILDLIFE AND DOMESTIC ANIMALS

Aquatic Life

Chlordane or oxychlordane residues have been found in fish; but other than this fact, little information was found in literature regarding toxicity of chlordane to aquatic life.

Plants

Chlordane or oxychlordane residues have been found in fish; but other than this fact, little information was found in literature regarding toxicity of chlordane to plants.

Invertebrates

Studies indicate that chlordane may produce toxic effects in certain soil invertebrates after surface application. Although little information concerning bioaccumulation in these organisms is available, the potential bioaccumulation of chlordane or oxychlordane by terrestrial insectivores is of concern.

Birds

Oral LD_{50} value for chlordane ranging from 331 to 858 ppm in the diet (approximately 25-50 mg/kg) are reported for a variety of wild bird species.

Mammals

Oral LD_{50} values ranging from 100 to 1,000 mg/kg are reported for a variety of animals, including rodents, goats, and sheep.

REGULATIONS AND RECOMMENDED GUIDELINES

Ambient Water Quality Criteria (USEPA, 1986):

Aquatic Life (Freshwater)

Acute toxicity: 2.4 ug/liter
Chronic toxicity: 0.0043 ug/liter

Aquatic Life (Saltwater)

Acute toxicity: 0.09 ug/liter
Chronic toxicity: 0.0040 ug/liter

Human Health

Due to the carcinogenicity of Chlordane, the ambient water criterion is set at zero. However, estimates of the carcinogenic risks due to ingestion of contaminated water and contaminated aquatic organisms are:

Risk	Concentration
10^{-4}	46 ng/liter
10^{-5}	4.6 ng/liter
10^{-6}	0.46 ng/liter
10^{-7}	0.046 ng/liter

National Primary Drinking Water Standard:

0.005 mg/liter (Proposed MCL; 50 Federal Register 46904, Wed., Nov. 13, 1985)

Department of Transportation:

Combustible liquid

OSHA PEL 29 CFR 1910.1000

TWA = 0.5 mg/m^3 (skin)

ACGIH TLV:

TWA = 0.5 mg/m^3 (skin)

DOSE-RESPONSE ASSESSMENT

The human dose-response parameter estimates for carcinogens and noncarcinogens are computed differently by USEPA; therefore, these estimates are presented separately below.

Carcinogenic Effects

The Cancer Assessment Group (CAG) of USEPA has derived an oral cancer potency estimate for chlordane of 1.3 $(mg/kg/day)^{-1}$ (USEPA, 1990b).

The CAG has also derived an inhalation cancer potency estimate of 1.3 $(mg/kg/day)^{-1}$ (USEPA, 1990b).

Oral Cancer Potency Estimate: 1.3 $(mg/kg/day)^{-1}$ (USEPA, 1990b)
Inhalation Cancer Potency Estimate: 1.3 $(mg/kg/day)^{-1}$ (USEPA, 1990b)

Noncarcinogenic Effects

The oral intake reference dose (RfD) for chlordane is 0.0006 mg/kg/day based on a study assessing the toxicity of dietary chlordane (1 ppm) in rats over a period of 130 weeks (USEPA, 1990a). The inhalation intake RfD has not been determined for chlordane at this time.

Oral RfD = 0.00006 mg/kg/day

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USEPA. 1990b. Integrated Risk Information System (IRIS). Access Date: June 29, 1990. [Note: This is a computerized database.]

CHLOROBENZENE

SUMMARY

Chlorobenzene (monochlorobenzene) is used as a solvent and as a raw material in chemical manufacturing. It is persistent in the environment and can be adsorbed to organic material in soil. Chlorobenzene may cause liver tumors in male rats. Animals exposed to chlorobenzene have exhibited liver and kidney damage.

CAS Number: 108-90-7

Chemical Formula: C6H5Cl

IUPAC Name: Chlorobenzene

Important Synonyms and Trade Names: Monochlorobenzene, benzene chloride, phenyl chloride

CHEMICAL AND PHYSICAL PROPERTIES

Molecular Weight: 112.6

Boiling Point: 131°C

Melting Point: -46°C

Specific Gravity: 1.11 at 20°C (liquid)

Solubility in Water: 500 mg/liter (Verschueren, 1977)
300 mg/liter (Tewari et al., 1982)
448 mg/liter (Mayo, 1980)
625 mg/liter (Valvani et al., 1980)

Solubility in Organics: Soluble in alcohol, benzene, chloroform, ether, and carbon tetrachloride

Log Octanol/Water Partition Coefficient (Kow):
2.84 (Valvani et al., 1980; Leo et al., 1971)
2.98 (Tewari et al., 1982)

Soil/Water Partition Coefficient (Koc):

125 (Sabljic, 1984) (Table 1)
836; 996 (Lyman et al., 1982) Eqn 4-8 (log Kow = 2.84; 2.98)
470; 604 (Lyman and Loreti, 1987) (log Kow = 2.84; 2.98)
330 (USEPA, 1986a)

Bioconcentration Factor:

24.8-16.4	(Davies and Dobbs, 1984) Eqn 3 (S = 300 - 625)
10.3	(USEPA, 1980)
84.8; 108.3	(Lyman et al., 1982) Eqn 5-2 (log Kow = 2.84; 2.98)
83	(Davies and Dobbs, 1984) Eqn B (log Kow = 2.9)
41	(Davies and Dobbs, 1984) Eqn C (log Kow = 2.9)
94	(Lyman et al., 1982) Eqn 5-2 (log Kow = 2.9)

Vapor Pressure: 8.8 mm Hg at 20°C (TDB Peer Review Committee, 1984)
10 mm Hg at 22.2°C (Perry and Chilton, 1973)
20 mm Hg at 35.3°C (Perry and Chilton, 1973)
11.7 mm Hg (USEPA, 1986a)

Vapor Density: 3.88

Henry's Law Constant: 3.56×10^{-3} atm-m^3/mole at 25°C (calculated)
1.56×10^{-1} Dimensionless

Flash Point: 28°C

TRANSPORT AND FATE

Chlorobenzene is removed from surface water primarily by volatilization. Following emission to the air, chlorobenzene is likely to degrade slowly through chemical and photolytic reactions. A range of experimental and estimated Kocs is reported above and indicates that sorption of chlorobenzene to soils/sediments and dissolved organic material will occur. Pavlou (1980) estimates that sorption of volatile organic compounds will range from low to moderate. The combined water solubility and moderate organic partitioning suggest that chlorobenzene will exhibit some degree of environmental mobility.

A range of BCFs for chlorobenzene is also presented above. ASTM (1985) indicates that chemicals with BFCs less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors suggests that appreciable bioconcentration or biomagnification of chlorobenzene residues is not likely to occur.

HEALTH EFFECTS

A study of the carcinogenicity of chlorobenzene was recently completed by the National Toxicology Program (NTP, 1986). Results show that chlorobenzene caused a statistically significant increase in neoplastic nodules in the livers of high-dose male rats but was not carcinogenic in female rats or in mice of either sex. However, there were also hepatocellular carcinomas in two vehicle control male rats and combining these with the neoplastic nodule data results in a borderline significance for high-dose males in at least one statistical test (USEPA, 1986b). Monochlorobenzene has been classified in USEPA's Group C according to USEPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon the preliminary data from the NTP study.

Increased mitotic crossovers (indicators of DNA damage) were observed in Saccharomyces cerevisiae exposed to monochlorobenzene (USEPA, 1985a). Additionally, monochlorobenzene induced reversions to vitamin B1 prototrophy in another study utilizing Streptomyces (USEPA, 1985a). Negative results were obtained in chromosome aberration tests utilizing Chinese hamster ovary cells; however, positive evidence of sister chromatid exchange was achieved in the same test medium (NTP, 1986).

Occupational studies suggest that chronic exposure to monochlorobenzene vapor may cause blood disorders, hyperlipidemia, and cardiac dysfunction in humans. Like many organic solvents, monochlorobenzene is a central nervous system depressant in exposed humans, but no chronic neurotoxic effects have been reported (USEPA, 1985a). It is also irritating to the eyes and respiratory tract (USEPA, 1986b). Animals exposed to chlorobenzene have exhibited liver and kidney damage. Dogs exposed to chlorobenzene vapors at doses of 2.0 mg/l for 6 hours/day, 5 days/week exhibited bilateral atrophy of the epithelial tissue in the seminiferous tubules (USEPA, 1985a). No studies on the teratogenicity of chlorobenzene were located in available literature. The oral LD_{50} value for chlorobenzene in rats was 2,910 mg/kg (NIOSH, 1982).

TOXICITY TO WILDLIFE AND DOMESTIC ANIMALS

Aquatic Life

Chlorobenzene was acutely toxic to fish at levels greater than 25 mg/l and to aquatic invertebrates at levels greater than 10 mg/l (USEPA, 1980). Data from chronic studies on the toxicity of chlorobenzene to aquatic life were not found in the literature reviewed.

Plants

Information was not found in the literature reviewed regarding toxicity of chlorobenzene to plants.

Invertebrates

Information was not found in the literature reviewed regarding toxicity of chlorobenzene to invertebrates.

Birds

Information was not found in the literature reviewed regarding toxicity of chlorobenzene to birds.

Mammals

Information was not found in the literature reviewed regarding toxicity of chlorobenzene to mammals.

REGULATIONS AND RECOMMENDED GUIDELINES

Ambient Water Quality Criteria (USEPA, 1986c):

The available data are not adequate for establishing freshwater or saltwater criteria. However, USEPA does report the lowest values known to cause toxicity in aquatic organisms.

Aquatic Life (Freshwater)

Acute toxicity: 250 ug/liter
Chronic toxicity: 50 ug/liter

Aquatic Life (Saltwater)

Acute toxicity: 160 ug/liter
Chronic toxicity: 129 ug/liter

Human Health

Health criterion: 488 ug/liter
Organoleptic criterion: 20 ug/liter

National Primary Drinking Water Standard (USEPA):

0.06 mg/liter (Proposed RMCL; 50 Federal Register 47001, Wednesday, November 13, 1985)

OSHA PEL 29 CFR 1910.1000

TWA = 75 ppm

ACGIH TLV

TWA = 75 ppm

DOSE-RESPONSE ASSESSMENT

The human dose-response parameter estimates for carcinogens and noncarcinogens are computed differently by USEPA; therefore, these estimates are presented separately below.

Carcinogenic Effects

No carcinogenic effects from chlorobenzene exposure have been documented in the literature reviewed.

Noncarcinogenic Effects

For chlorobenzene (monochlorobenzene), the oral intake rate is based on the reference dose (RfD) of 2×10^{-2} mg/kg/day reported in the USEPA Health Effects Assessment Summary Table (USEPA, 1990). It is a subchronic study in which dogs were administered doses of 27.3 mg/kg/day by capsule for 90 days. The endpoint of toxicological concern was liver and kidney effects. An uncertainty factor of 1,000 was incorporated by USEPA in the derivation of the RfD. Additional details on the underlying study were not available.

The inhalation intake rate for chlorobenzene is based on the inhalation RfD of 5 x 10^{-3} mg/kg/day reported in the USEPA HEAST (USEPA, 1990). The RfD is derived from a subchronic inhalation study in which rats were exposed to 75 ppm chlorobenzene (53 mg/kg/day) 7 hours/day, 5 days/week for 120 days (Dilley, 1977). Among the effects noted at this dose were small focal lesions in the adrenal cortex and kidney tubules and decreased SGOT. Therefore, 75 ppm constitutes a LOAEL. An uncertainty factor of 10,000 was incorporated in the derivation of the RfD by USEPA.

Oral RfD = 2 x 10^{-2} $(mg/kg/day)^{-1}$ (USEPA, 1990)
Inhalation RfD = 5 x 10^{-3} $(mg/kg/day)^{-1}$ (USEPA, 1990)

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CHLOROFORM

SUMMARY

Chloroform is an organic solvent which was used in the manufacture of azodrin and bidrin at RMA. Chloroform (trichloromethane) is often produced during the chlorination of drinking water and thus is a common drinking water contaminant. It is volatile in surface waters and is not likely to be persistent in the environment. Chloroform causes an increase of kidney epithelial tumors in rats and of hepatocellular carcinomas in mice. In addition, there is suggestive evidence from epidemiological studies that exposure to chloroform and other trihalomethanes is associated with an increased incidence of bladder tumors in humans. Other toxic effects of chloroform include central nervous system depression; eye, skin, and gastrointestinal irritation; and damage to the liver, heart, and kidney.

CAS Number: 67-66-3

Chemical Formula: $CHCl_3$

IUPAC Name: Trichloromethane

CHEMICAL AND PHYSICAL PROPERTIES

Molecular Weight: 119.38

Boiling Point: 61.7°C

Melting Point: -63.5°C

Specific Gravity: 1.4832 at 20°C

Solubility in Water: 8,200 mg/liter at 20°C
7,500 mg/liter at 20°C (Valvani et al., 1980)
9,200 mg/liter at 25°C (Davies and Dobbs, 1984)

Solubility in Organics: Soluble in acetone; miscible with alcohol, ether, and benzene

Log Octanol/Water Partition Coefficient (Kow):

1.97	(Moriguchi, 1975)
1.90	(Davies and Dobbs, 1984)
1.96	(Valvani et al., 1980)

Soil-Water Partition Coefficient (Koc):

45	(Sabljic, 1984)
257; 281	(Lyman et al., 1982) Eqn 4-8 (log Kow = 1.9; 1.97)
87; 99	(Lyman and Loreti, 1987) (log Kow = 1.90; 1.97)

Bioconcentration Factor (BCF):

16	(Lyman et al., 1982) Eqn 5-2 (log Kow = 1.9)
18.18	(Lyman et al., 1982) Eqn 5-2 (log Kow = 1.96)
3.59; 4.03	(Davies and Dobbs, 1984) Eqn A (S = 7,500, 9,200)
21	(Davies and Dobbs, 1984) Eqn B (log Kow = 1.9)
12	(Davies and Dobbs, 1984) Eqn C (log Kow = 1.9)

Vapor Pressure: 150.5 mm Hg at 20°C
200 mm Hg at 25.9°C (Perry and Chilton, 1973)

Vapor Density: 4.12

Henry's Law Constant: 3.9×10^{-3} atm-m^3/mole (calculated)
2.87×10^{-3} atm-m^3/mole (USEPA, 1985a)
1.21×10^{-1} Dimensionless

TRANSPORT AND FATE

Due to its high vapor pressure, volatilization is the major transport process for removal of chloroform from aquatic systems (USEPA, 1980). Once in the troposphere, chloroform is attacked by hydroxyl radicals with the subsequent formation of phosgene (COCl2) and possibly chlorine oxide (ClO) radicals. Neither of these reaction products is likely to persist; phosgene is readily hydrolyzed to hydrochloric acid and carbon dioxide. Reaction with hydroxyl radicals is thought to be the primary environmental fate of chloroform. However, chloroform that remains in the troposphere may return to earth in precipitation or be adsorbed on particulates, and a small amount may diffuse upward to the stratosphere where it photodissociates via interaction with ultraviolet light (USEPA, 1985b). Neither photolysis nor hydrolysis appear to be significant environmental fate processes for chloroform (USEPA, 1985b).

A range of estimated Kocs is reported above and indicates that sorption of chloroform to soils/sediments and dissolved organic material will occur. Pavlou (1980) estimates that sorption of volatile organic compounds will range from low to moderate. The combined high

water solubility and low organic partitioning of chloroform suggest that this compound will exhibit a high degree of environmental mobility.

Studies with marine organisms provide evidence for only weak to moderate bioaccumulation of chloroform. A range of estimated BCFs for chloroform is also presented above. ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up foodchains. The magnitude of the concentration factors suggests that appreciable bioconcentration or biomagnification of chloroform residues is not likely to occur.

HEALTH EFFECTS

Humans may be exposed to chloroform by inhalation, ingestion, or skin contact. Epidemiological studies suggest that higher concentrations of chloroform and other trihalomethanes in water supplies may be associated with an increased frequency of bladder cancer in humans. Other toxic effects include local irritation of the skin or eyes, central nervous system depression, gastrointestinal irritation, liver and kidney damage, arrhythmia, tachycardia, and bradycardia. Death from chloroform can occur at high dose levels and is attributed to ventricular fibrillation. Chloroform anesthesia can produce delayed death as a result of liver necrosis.

In laboratory animals, exposure to chloroform by inhalation, intragastric administration, or intraperitoneal injection produces liver and kidney damage. Chronic administration of chloroform by gavage is reported to produce a dose-related increase in the incidence of kidney epithelial tumors in rats and a dose-related increase in the incidence of hepatocellular carcinomas in mice (IARC, 1979; USEPA, 1980). Based on USEPA's Proposed Carcinogen Risk Assessment Guidelines, chloroform is classified in USEPA's Group B2 (probable human carcinogen) based upon sufficient evidence of carcinogenicity in animals and inadequate epidemiologic evidence (USEPA, 1985b).

An increased incidence of fetal abnormalities was reported in offspring of pregnant rats exposed to chloroform by inhalation. Oral doses of chloroform that caused maternal toxicity

produced relatively mild fetal toxicity in the form of reduced birth weights. There are limited data suggesting that chloroform has mutagenic activity in some test systems. However, negative results have been reported for bacterial mutagenesis assays.

The oral LD_{50} and inhalation LC_{LO} values for chloroform in the rat are 908 mg/kg and 39,000 mg/m^3 per 4 hours, respectively (ACGIH, 1980).

TOXICITY TO WILDLIFE AND DOMESTIC ANIMALS

Aquatic Life

Limited information is available concerning toxicity of chloroform to organisms exposed at known concentrations (USEPA, 1980). Median effect concentrations for two freshwater and one invertebrate species range from 28,900 to 115,000 µg/l. Twenty-seven day LC_{50} values of 2,030 and 1,240 µg/l were reported for embryo-larval tests with rainbow trout in water at two levels of hardness. The only reliable result concerning the toxicity of chloroform to saltwater life is a 96-hour LC_{50} value of 1,500 µg/l for pink shrimp.

Plants

Information was not found in the literature reviewed regarding toxicity of chloroform to plants.

Invertebrates

Information was not found in the literature reviewed regarding toxicity of chloroform to invertebrates.

Birds

Information was not found in the literature reviewed regarding toxicity of chloroform to birds.

Mammals

No data were located on the toxicity of chloroform to wild or domestic animals in the literature reviewed. Conceivably, acute effects on wildlife can occur in the vicinity of a major chloroform spill; however, chronic effects from long-term exposure to low ambient levels is unlikely (USEPA, 1985b).

REGULATIONS AND RECOMMENDED GUIDELINES

Ambient Water Quality Criteria (USEPA, 1986):

The available data are not adequate for establishing criteria. However, USEPA does report the lowest values known to be toxic in freshwater aquatic organisms.

Aquatic Life (Freshwater)

Acute Toxicity: 28,900 µg/liter
Chronic Toxicity: 1,240 µg/liter

Human Health

Due to the carcinogenicity of chloroform the ambient water criterion is set at zero. However, estimates of the carcinogenic risks associated with lifetime exposure from the ingestion of contaminated water and contaminated aquatic organisms are:

Risk	Concentration
10^{-4}	19.0 µg/liter
10^{-5}	1.90 µg/liter
10^{-6}	0.19 µg/liter
10^{-7}	0.019 µg/liter

NIOSH REL:

Ceiling = 2 ppm (1 hr)

OSHA PEL 29 CFR 1910.1000

TWA = 2 ppm

ACGIH TLV:

TWA = 10 ppm

DOSE-RESPONSE ASSESSMENT

The human dose-response parameter estimates for carcinogens and noncarcinogens are computed differently by USEPA, therefore, these estimates are presented separately below.

Carcinogenic Effects

The Cancer Assessment Group (CAG) of USEPA has derived an oral cancer potency estimate for chloroform of 6.1×10^{-3} $(mg/kg/day)^{-1}$ (USEPA, 1990b).

The CAG has also derived an inhalation cancer potency estimate of 8.1×10^{-3} $(mg/kg/day)^{-1}$ (USEPA, 1990b).

Oral Cancer Potency Estimate: 6.1×10^{-3} $(mg/kg/day)^{-1}$ (USEPA, 1990b).
Inhalation Cancer Potency Estimate: 8.1×10^{-3} $(mg/kg/day)^{-1}$ (USEPA, 1990b).

Noncarcinogenic Effects

The oral intake reference dose (RfD) for chloroform is 0.01 mg/kg/day based on a study assesisng the toxicity of dietary chloroform in dogs dosed with 15 mg/kg 6 days/week over a period of 7.5 years (USEPA, 1990a). The inhalation intake RfD has not been determined for chloroform at this time.

Oral RfD = 0.01 mg/kg/day

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CHLOROPHENYLMETHYL SULFIDE

SUMMARY

This compound is a process intermediate which was formed during the manufacture of Planavin at RMA. Data regarding the toxicity of chlorophenylmethyl sulfide (CPMS) to humans was not located in literature reviewed. Rats exposed subchronically (91 days) to oral doses of CPMS at or above the maximum tolerated dose (i.e., 750 ppm) experienced elevated serum potassium and calcium levels, reduced serum glutamic oxalate transaminase (SGOT) levels, and red blood cell counts. Rhesus monkeys subacutely exposed to oral doses of CPMS experienced mortality at the highest dose. At lower doses increased BUN, liver and kidney weights, and decreased serum alkaline phosphatase were observed. Liver lesions were observed at all dose levels. No effects were observed in mice or rats fed CPMS at 281 ppm for a period of 28 days. CPMS tested negative in the Ames mutagenicity assay.

CAS Number: 123-09-1

Chemical Formula: C_7H_7SCl

IUPAC Name: Chlorophenylmethyl Sulfide

Important Synonyms and Trade Names: CPMS, p-chlorothioanisole

CHEMICAL AND PHYSICAL PROPERTIES

Molecular Weight: 158.7

Melting Point: 17-19°C (Miller et al., 1976)

Boiling Point: 220-224°C (Miller et al., 1976)

Solubility in Water: 12 mg/liter (estimated) (Lyman et al., 1982)

Specific Gravity: 1.2 at 49°C (Miller et al., 1976)

Vapor Pressure: 20 mm Hg at 118°C (Miller et al., 1976)
0.11 mm Hg at 25°C (estimated) (Lyman et al., 1982)
0.05 mm Hg (Lyman et al., 1982) Method 1

Henry's Law Constant: 1.9×10^{-3} atm-m^3/mole (calculated)
8.35×10^{-4} atm-m^3/mole (calculated)
2.7×10^{-2} Dimensionless

Log Octanol/Water Partition Coefficient (Kow): 3.22 (Brueggemann, 1982)

Soil to Water Partition Coefficient (Koc):

1,345 (Lyman et al., 1982) Eqn 4-8 (log Kow = 3.22)
930 (Lyman and Loreti, 1987) (log Kow = 3.22)

Bioconcentration Factor:

69.1 (Davies and Dobbs, 1984) Eqn C (log Kow = 3.35)
154 (Davies and Dobbs, 1984) Eqn B (log Kow = 3.35)
207 (Lyman et al., 1982) Eqn 5-2 (log Kow = 3.35)

TRANSPORT AND FATE

The low to moderate vapor pressure of CPMS indicates that some volatilization (evaporation) from soil surfaces would be expected to occur. CPMS is relatively insoluble in water (Cogley and Foy, 1978). A range of estimated Kocs is reported above and indicates that sorption of CPMS to soils/sediments and dissolved organic material will occur. The combined low water solubility and high organic partitioning indicates that little environmental mobility would be expected for this compound.

Microbial degradation reportedly occurs (Cogley and Foy, 1978); however, the extent of degradation and the resulting degradation intermediates were not reported. CPMS can be chemically oxidized to sulfoxide under relatively mild conditions. The persistence (half-life) of CPMS in soil may vary from one to six months depending on ambient conditions (Cogley and Foy, 1978). Guenzi et al. (1979) reported that 61 percent of soil applied CPMS was retained for 160 days.

Data on the uptake of CPMS in various plant parts has been reported (Guenzi et al., 1979). At a soil concentration of 0.35 mg/g soil, concentration factors for tops and roots, respectively, of various plants ranged from 15 and 6 in corn to 64 and 6 in sugarbeets.

A range of estimated BCFs for CPMS is also presented above. ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains.

The magnitude of the concentration factors suggests that bioconcentration or biomagnification of CPMS residues may occur.

HEALTH EFFECTS

No data on the toxicity of CPMS in humans was located in the reviewed literature. Pharmacokinetic (Thake et al., 1979) and metabolic data (Menn et al., 1975; Oehler and Ivie, 1983) indicate that the sulfide is converted predominantly to the sulfone in vivo and excreted predominantly in the urine.

In a 28-day feeding study (Thake et al., 1979) both mice and rats were administered CPMS in their feed at concentrations of 281, 562, 2,250, and 4,500 ppm (mice) or 281, 562, 2,250, and 9,000 ppm (rats). All of the rats in the highest dose group exhibited a pronounced taste aversion, associated anorexia, and subsequent mortality due to starvation. At doses greater than 281 ppm there was a reduction in weight gain of rats and reduced food consumption. No effects on body weight gain in mice were observed at the lower dose levels. The NOEL identified from this study was 281 ppm.

Rats and mice were exposed subchronically (91 days) to CPMS at 750, 1,500, and 3,000 ppm. Rats were more sensitive than mice and experienced reduced red blood cell counts; males experienced reduced SGOT levels (Thake et al., 1979). Serum potassium and calcium levels were markedly elevated in both males and females, and increased liver and kidney weights were observed. Compound-related lesions were present in the livers of exposed rats. Mutagenicity tests utilizing the Ames (Salmonella) assay were negative (Thake et al., 1979).

Subacute oral toxicity studies (14 days) employing dosages of 5, 10, and 20 mg/kg in Rhesus monkeys resulted in mortalities at the highest dose (Thake et al., 1979). The clinical signs of toxicity included emesis, anorexia, hypothermia, depression, weakness, and diarrhea. Thrombocytopenia and neutrophilia were apparent at the two lower dosages. Increased BUN, decreased serum alkaline phosphatase, and increased liver and kidney weights were observed at the 10 mg/kg dose. Liver lesions consisting of vacuolization of hepatocytes and necrosis

were observed at low dosages. At higher dosages, vacuolization of proximal tubular epithelium was observed. However, the general poor health of the animals used in this study (i.e., treatment and control) precludes any meaningful interpretation of the data.

Acute oral toxicity of CPMS in rats ranged from 479 (female) to 619 mg/kg (male). Acute oral toxicity in mice ranged from 672 (female) to 877 mg/kg. Clinical signs included dyspnea (labored breathing) and lacrimation (Thake et al., 1979). Dermal LD_{50} values in rats ranged from 2,190 to 5,630 mg/kg.

TOXICITY TO WILDLIFE AND DOMESTIC ANIMALS

Aquatic Life

Information was not found in the literature reviewed regarding toxicity of CPMS to aquatic life.

Plants

Information was not found in the literature reviewed regarding toxicity of CPMS to plants.

Invertebrates

Information was not found in the literature reviewed regarding toxicity of CPMS to invertebrates.

Birds

Information was not found in the literature reviewed regarding toxicity of CPMS to birds.

Mammals

Information was not found in the literature reviewed regarding toxicity of CPMS to mammals.

REGULATIONS AND RECOMMENDED GUIDELINES

None.

DOSE-RESPONSE ASSESSMENT

The human dose-response parameter estimates for carcinogens and noncarcinogens are computed differently by USEPA; therefore, these estimates are presented separately below.

Carcinogenic Effects

USEPA has not developed oral or inhalation cancer potency estimates for CPMS.

Noncarcinogenic Effects

The USEPA has not currently derived a reference dose (RfD) for CPMS.

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CHLOROPHENYLMETHYL SULFONE

SUMMARY

Chlorophenylmethyl sulfone is a process intermediate which was formed during the manufacture of Planavin at RMA. Data on the toxicity of chlorophenylmethyl sulfone ($CPMSO_2$) in humans were not located in literature reviewed. $CPMSO_2$ was mildly irritating to the skin of rabbits following dermal application. Subchronic exposures to $CPMSO_2$ in rats at or above the maximally tolerated dose (i.e., 750 ppm) resulted in altered blood serum chemistry, liver lesions, and increased liver and kidney weights. In monkeys, subacute oral exposures to $CPMSO_2$ resulted in mortality at the highest doses, and increased levels of BUN, serum enzymes, and sodium at all other dose levels. No effect was observed in rats fed $CPMSO_2$ at a concentration of 281 ppm for 28 days. $CPMSO_2$ was not mutagenic when evaluated using the Ames Salmonella assay.

CAS Number: 98-57-7

Chemical Formula: $C_7H_7SO_2Cl$

IUPAC Name: Chlorophenylmethyl Sulfone

Important Synonyms and Trade Names: $CPMSO_2$, 1-chloro, 4-methylsulfoxylbenzene

CHEMICAL AND PHYSICAL PROPERTIES

Molecular Weight: 190.6

Melting Point: 92-99°C (Miller et al., 1976)

Solubility in Water: 1,050 mg/liter (estimated) (Lyman et al., 1982)
1,170 mg/liter (estimated) (Lyman et al., 1982)

Vapor Pressure: 5×10^{-4} mm Hg at 25°C (estimated) (Lyman et al., 1982)
2.5 mm Hg at 132°C (Miller et al., 1976)
0.003 (Lyman et al., 1982) (Method 1)

Henry's Law Constant: 1.2×10^{-7} atm-m^3/mole (calculated)
4.29×10^{-6} atm-m^3/mole (calculated)
3.5×10^{-4} Dimensionless

Log Octanol/Water Partition Coefficient (Kow): 1.20 (Brueggemann, 1982)

Soil to Water Partition Coefficient (Koc):

126 (Lyman et al., 1982) Eqn 4-8 (log Kow = 1.33)
31 (Lyman and Loreti, 1987) (log Kow = 1.33)

Bioconcentration Factor:

5.54 (Davies and Dobbs, 1984) Eqn C (log Kow = 1.21)
8.14 (Davies and Dobbs, 1984) Eqn B (log Kow = 1.21)
4.89 (Lyman et al., 1982) Eqn 5-2 (log Kow = 1.21)

TRANSPORT AND FATE

The low vapor pressure of $CPMSO_2$ indicates that volatilization (evaporation) from soil/water surfaces is not likely to be a major transport process for this chemical. $CPMSO_2$ is low to moderately soluble in water (Cogley and Foy, 1978). A range of estimated Kocs is reported above and indicates that some sorption of $CPMSO_2$ to soils/sediments and dissolved organic material may occur. The combined water solubility and low organic partitioning indicate that $CPMSO_2$ will exhibit some degree of environmental mobility. Microbial degradation reportedly occurs (Cogley and Foy, 1978); however, the extent to which $CPMSO_2$ is utilized and the resulting degradation products were not reported.

The persistence (half-life) of $CPMSO_2$ in soil may vary from 6 months to 1 year depending on ambient conditions (Cogley and Foy, 1978). Guenzi et al. (1979) reported that 82.5 percent of applied sulfone in soil (4.77 mg/g) incubated at 30°C was retained following 160 days. Uptake of sulfone in selected plants was also reported by Guenzi et al (1979). Concentration factors in the tops and roots ranged from 19 and 6, respectively, in corn to 72 and 5 in sugar beets.

A range of estimated BCFs for $CPMSO_2$ is also reported above. ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors suggests that appreciable bioconcentration or biomagnification of $CPMSO_2$ residues is not likely to occur.

HEALTH EFFECTS

No data on the toxicity of $CPMSO_2$ in humans were located in available literature. Sulfone was nonmutagenic utilizing the Ames assay (Thake et al., 1979). Data on the carcinogenicity of $CPMSO_2$ were not located in available literature.

Topical application of $CPMSO_2$ to the skin of rabbits produced mild irritation and no effect was noted following ocular treatment (Thake et al., 1979). Pharmacokinetic (Thake et al., 1979) and metabolic data (Menn et al., 1975; Oehler and Ivie, 1983) indicate that conjugated metabolites are excreted predominantly in the urine and largely as the sulfone. In a 28-day feeding study (Thake et al., 1979) both mice and rats were administered $CPMSO_2$ in their feed at concentrations of 281, 562, 2,250, and 4,500 ppm (mice) or 281, 562, 2,250, and 9,000 ppm (rats). At doses greater than 281 ppm rats exhibited a decrease in weight gain, and reduced food consumption was also noted. No other clinical signs were observed. No effects on weight gain were observed in treated mice at lower dose levels. The NOEL from the rat study (i.e., the more sensitive species) was 281 ppm.

Rats exposed subchronically (91 days) to $CPMSO_2$ at concentrations of 750, (maximum tolerated dose), 1,500 and 3,000 ppm in the diet experienced reduced red blood cell counts, and males had elevated BUN levels (Thake et al., 1979). Males and females experienced elevated serum potassium and calcium, and liver and kidney weights were markedly increased. Compound-related lesions were present in the livers of rats of both sexes. Post-mortem necropsies of sacrificed animals indicated incipient tumor formation.

Subacute oral toxicity studies (14 days) were conducted with dosages of 2.5, 5, 10, 15, 20, and 30 mg/kg $CPMSO_2$ in Rhesus monkeys (Thake et al., 1979). Doses of 20 and 30 mg/kg were lethal. Clinical signs included anorexia, emesis, and diarrhea. Increased BUN, SGOT, and sodium values as well as decreased serum glucose and inorganic phosphorus were observed at 10 mg/kg and higher doses. Increased adrenal weights were also noted at this dose level. Lymphoid tissue hyperplasia was noted as were liver lesions. However, the general poor health of the animals used in this study (i.e., treatment and control) precludes any meaningful interpretation of the data.

TOXICITY TO WILDLIFE AND DOMESTIC ANIMALS

Aquatic Life

Information was not found in the literature reviewed regarding toxicity of $CPMSO_2$ to aquatic life.

Plants

Information was not found in the literature reviewed regarding toxicity of $CPMSO_2$ to plants.

Invertebrates

Information was not found in the literature reviewed regarding toxicity of $CPMSO_2$ to invertebrates.

Birds

Information was not found in the literature reviewed reagarding toxicity of $CMPSO_2$ to birds.

Mammals

Information was not found in the literature reviewed regarding toxicity of $CMPSO_2$ to mammals.

REGULATIONS AND RECOMMENDED GUIDELINES

None.

DOSE-RESPONSE ASSESSMENT

The human dose-response parameter estimates for carcinogens and noncarcinogens are computed differently by USEPA; therefore, these estimates are presented separately below.

Carcinogenic Effects

USEPA has not developed oral or inhalation cancer potency estimates for $CPMSO_2$.

Noncarcinogenic Effects

The USEPA has not currently derived a reference dose (RfD) for $CPMSO_2$.

REFERENCES

ASTM (American Society of Testing and Materials). 1985. Standard Practice for Conducting Bioconcentration Tests with Fishes and Saltwater Bivalve Mollusca. Designation E 1022-84, pages 590-62. In: 1985 Annual Book of ASTM Standards Volume 11.04. American Society for Testing and Materials, Philadelphia, Pennsylvania.

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CHLOROPHENYLMETHYL SULFOXIDE

SUMMARY

No data regarding the toxicity of chlorophenylmethyl sulfoxide (CPMSO) to humans were located in available literature. Rats chronically exposed to CPMSO at or above the maximum tolerated dose (i.e., 750 ppm) experienced reduced red blood cell counts, reduced levels of certain serum enzymes, emaciation, increased liver and kidney weights, liver lesions, and mortality. Subacute dosages of CPMSO to Rhesus monkeys resulted in depression, anorexia, emesis, hypothermia, decreased red blood cell count, increased levels of serum enzymes, nitrogen wastes, and calcium levels, and increased kidney and liver weights. No data on the carcinogenicity of CPMSO were located in the available literature. Mutagenicity assays using Salmonella were negative.

CAS Number: 934-73-6

Chemical Formula: C_7H_7SOCl

IUPAC Name: Chlorophenylmethyl Sulfoxide

Important Synonyms and Trade Names: CPMSO, 1-chloro, 4-methylsulfinylbenzene

CHEMICAL AND PHYSICAL PROPERTIES

Molecular Weight: 174.6

Melting Point: 37-47°C (Miller et al., 1976)

Solubility in Water: 1,200 mg/liter (estimated) (Lyman et al., 1982)
1,050 mg/liter (estimated) (Lyman et al., 1982)

Vapor Pressure: 8×10^{-4} mm Hg at 25°C (estimated) (Lyman et al., 1982)
2.5 mm Hg at 131.5°C (Miller et al., 1976)
0.078 (Lyman et al., 1982) Method 1

Henry's Law Constant: 1.5×10^{-7} atm-m^3/mole (calculated)
6.3×10^{-4} Dimensionless

Log Octanol/Water Partition Coefficient (Kow): 1.33 (Brueggemann, 1982)
1.26 (Lyman et al., 1982) Fragment Method

Soil to Water Partition Coefficient (Koc):

107 (Lyman et al., 1982) Eqn 4-8 (log Kow = 1.20)
25 (Lyman and Loreti, 1987) (log Kow = 1.20)

Bioconcentration Factor:

5.88 (Davies and Dobbs, 1984) Eqn C (log Kow = 1.26)
8.71 (Davies and Dobbs, 1984) Eqn B (log Kow = 1.26)
5.34 (Lyman et al., 1982) Eqn 5-2 (log Kow = 1.26)

TRANSPORT AND FATE

The low vapor pressure of CPMSO indicates that volatilization (evaporation) from soil/water surfaces is not likely to be significant for this chemical. CPMSO is low to moderately soluble in water (Cogley and Foy, 1978). A range of estimated Kocs is reported above and indicates that some sorption of CPMSO to soils/sediments and dissolved organic material may occur. The combined water solubility and low organic partitioning suggest that CPMSO will exhibit some degree of environmental mobility.

Microbial degradation of CPMSO reportedly occurs (Cogley and Foy, 1978); however, the extent to which is utilized and the resulting degradation products were not reported. The persistence (half-life) of CPMSO in soil may vary from 6 months to 1 year depending on ambient conditions (Cogley and Foy, 1978). Guenzi et al. (1979) reported that 84.5 percent of applied sulfoxide in soil (4.77 ug/g) incubated at 30°C was retained following 160 days. Uptake of sulfoxide in selected plants was also reported by Guenzi et al. Concentration factors in the tops and roots ranged from 16 and 5, respectively, in corn, to 66 and 6 in sugarbeets.

A range of estimated BCFs is also reported above. ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors suggests that appreciable bioconcentration or biomagnification of CPMSO residues is not likely to occur.

HEALTH EFFECTS

No data on the toxicity of CPMSO in humans were located in available literature. CPMSO was nonmutagenic utilizing the Ames assay (Thake et al., 1979). Data on the carcinogenicity of CPMSO were not available.

CPMSO topical treatment produced mild skin irritation in rabbits and reversible lesions of the iris and conjunctival and corneal opacity when applied to the eye (Thake et al., 1979). Pharmacokinetic (Thake et al., 1979) and metabolic data (Menn et al., 1975; Oehler and Ivie, 1983) indicate that the conjugated metabolites are excreted predominantly in the urine and largely as the sulfone.

In a 28-day feeding study (Thake et al., 1979) both mice and rats were administered CPMSO in their feed at concentrations of 188, 375, 750, 1,300, 2,600, and 5,200 ppm (mice) or 162, 325, 650, 1,300, 2,600, and 5,200 ppm (rats). The highest dose in rats resulted in a significantly reduced feed consumption and mortality in 60 percent of the females and 80 percent of males. Feed consumption was unaffected at doses up to 1,300 ppm; however, weight gain was reduced in the 650 and 1,300 ppm dose groups. In the mouse study, two of five females in the high dose group died. Clinical signs included reduced body weight and reduced appetite prior to death. Locomotor activity was also depressed in these mice prior to death. Effects on body weight gain were observed in male mice at dose levels of 750 ppm and above. The NOEL from the rat study (i.e., the more sensitive species) was 325 ppm.

Rats exposed subchronically (91 days) to CPMSO in their food experienced reduced red blood cell counts, and males experienced reduced SGOT levels (Thake et al., 1979). Rats at the highest treatment were emaciated, and some mortalities occurred. Increased serum mineral levels and increased liver and kidney weights were also observed. Compound-related liver lesions were present in all dose groups. Post-mortem necropsies of sacrificed animals indicated incipient tumor formation.

Subacute oral toxicity studies (14 days) were conducted with dosages of 5, 10, and 20 mg/kg CPMSO in Rhesus monkeys (Thake et al., 1979). The highest dose produced depression,

anorexia, emesis, hypothermia, and weakness. Clinical signs were also observed at the lowest dose. Decreased red blood cell count occurred at 20 mg/kg, and increases in BUN, SGOT, serum glutamate pyruvate transaminase (SGPT), and calcium occurred at the two higher dosages. Increased liver and kidney weights were also observed at these dosages. Lymphoid tissue hyperplasia was observed at all dosages. However, the general poor health of the animals used in this study (i.e., treatment and control) precludes any meaningful interpretation of the data.

TOXICITY TO WILDLIFE AND DOMESTIC ANIMALS

Aquatic Life

Information was not found in the literature reviewed regarding toxicity of CPMSO to aquatic life.

Plants

Information was not found in the literature reviewed regarding toxicity of CPMSO to plants.

Invertebrates

Information was not found in the literature reviewed regarding toxicity of CPMSO to invertebrates.

Birds

Information was not found in the literature reviewed regarding toxicity of CPMSO to birds.

Mammals

Information was not found in the literature reviewed regarding toxicity of CPMSO to mammals.

REGULATIONS AND RECOMMENDED GUIDELINES

None.

DOSE-RESPONSE ASSESSMENT

The human dose-response parameter estimates for carcinogens and noncarcinogens are computed differently by USEPA; therefore, these estimates are presented separately below.

Carcinogenic Effects

USEPA has not developed oral or inhalation cancer potency estimates for CPMSO.

Noncarcinogenic Effects

The USEPA has not currently derived a reference dose (RfD) for CPMSO.

REFERENCES

ASTM (American Society of Testing and Materials). 1985. Standard Practice for Conducting Bioconcentration Tests with Fishes and Saltwater Bivalve Mollusca. Designation E 1022-84, pages 590-62. In: 1985 Annual Book of ASTM Standards Volume 11.04. American Society for Testing and Materials, Philadelphia, Pennsylvania.

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COPPER

SUMMARY

Copper is a mobile metal in the environment. Copper exists in a valence state of +1 or +2 and is found in nature as sulfide, oxide, or carbonate ore. It is toxic to humans at high levels and causes irritation following acute exposure and anemia following chronic exposure. Copper does not have teratogenic, mutagenic or carcinogenic effects in animals or humans. Sheep are very susceptible to copper toxicosis through the administration of worming medications. Copper appears to be toxic to some aquatic organisms at low water concentrations.

CAS Number: 7440-50-8

Chemical Formula: Cu

IUPAC Name: Copper

CHEMICAL AND PHYSICAL PROPERTIES

Atomic Weight: 63.546

Boiling Point: 2,567°C

Melting Point: 1,083°C

Specific Gravity: 8.92

Solubility in Water: Most copper salts are insoluble, with the exception of $CuSO_4$, $Cu(No_3)_2$, and $CuCl_2$ (the more common copper salts). The metal is insoluble in water.

Vapor Pressure: 1 mm Hg at 1,628°C

TRANSPORT AND FATE

Copper has two oxidation states, +1 (cuprous) and +2 (cupric). Cuprous copper is unstable in aerated water over the pH range of most natural waters (six to eight) and oxidizes to the cupric state. Several processes determine the fate of copper in the aquatic environment: formation of complexes, especially with humic substances; sorption to hydrous metal oxides, clays, and organic materials; and bioaccumulation. In waters polluted with soluble organic material, complexation with organic ligands can occur, thus favoring the prolonged dispersion

of copper in solution. The presence of organic acids also can lead to the mobilization of copper from the sediments to solution.

Copper has a strong affinity for hydrous iron and manganese oxides, clays, carbonate minerals, and organic matter. Sorption to these materials, both suspended in the water column and in the sediment, results in relative enrichment of the solid phase and reduction in dissolved level. Sorption processes are quite efficient in scavenging dissolved copper and in controlling its mobility in natural unpolluted streams. The amounts of the various copper compounds and complexes that actually exist in solution depend on the pH, temperature, alkalinity, and concentration of other chemical species. The levels of copper able to remain in solution are directly dependent on water chemistry. Generally, ionic copper is more soluble in low pH waters and less soluble in high pH waters.

As an essential nutrient, copper is accumulated by plants and animals, although it is not generally biomagnified. Because copper is strongly bioaccumulated and because biogenic ligands play an important role in complexing copper, biological activity is a major factor in determining the distribution and occurrence of copper in the ecosystem.

Bioconcentration factors in freshwater species range from zero for the bluegill to 2,000 for the alga Chlorella regularis. Among saltwater species, the highest bioaccumulation factors are those for the bivalve mollusks. Oysters can bioaccumulate copper up to 28,200 times without any significant mortality.

Because many copper compounds and complexes are readily soluble, copper is among the more mobile heavy metals in soil and other surface environments. The major process that limits the environmental mobility of copper is adsorption to organic matter, clays, and other materials. Atmospheric transport of copper compounds can also occur.

HEALTH EFFECTS

Copper appears to increase the mutagenic activity of triose reductone and ascorbic acid in bacterial test systems. However, copper itself does not appear to have mutagenic, teratogenic,

or carcinogenic effects in animals or humans. Copper has been classified in USEPA Group D, according to USEPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon inadequate evidence of carcinogenicity in both animals and humans (50 Federal Register 46968, Wednesday, November 13, 1985).

Dietary levels of trace elements such as molybdenum, sulfur, zinc, and iron can affect the level of copper that produces certain deficiency or toxicity symptoms. In general, more attention is given to the problems associated with copper deficiency than to problems of excess copper in the environment. However, high levels of copper can be toxic to humans.

Exposure to metallic copper dust can cause a short-term illness similar to metal fume fever that is characterized by chills, fever, aching muscles, dryness of mouth and throat, and headache. Exposure to copper fumes can produce upper respiratory tract irritation, a metallic or sweet taste, nausea, metal fume fever, and sometimes discoloration of skin and hair. Individuals exposed to dusts and mists of copper salts may exhibit congestion of nasal mucous membranes, sometimes of the pharynx, and occasionally ulceration with perforation of the nasal septum.

If sufficient concentrations of copper salts reach the gastrointestinal tract, they act as irritants and can produce salivation, nausea, vomiting, gastritis, and diarrhea. Elimination of ingested ionic copper by vomiting and diarrhea generally protects the patient from more serious systemic toxic effects, which can include hemolysis, hepatic necrosis, gastrointestinal bleeding, oliguria, azotemia, hemoglobinuria, hematuria, proteinuria, hypotension, tachycardia, convulsions, and death. Chronic exposure may result in anemia.

Copper salts act as skin irritants producing an itching eczema. Conjunctivitis or even ulceration and turbidity of the cornea may result from direct contact of ionic copper with the eye.

TOXICITY TO WILDLIFE AND DOMESTIC ANIMALS

Aquatic Life

Mean acute toxicity values for a large number of freshwater animals range from 7.2 mg/l for Daphnia pulicaria to 10,200 mg/l for the bluegill (USEPA, 1980). Toxicity tends to decrease as hardness, alkalinity, and total organic carbon increase. Chronic values for a variety of freshwater species range from 3.9 mg/l for brook trout to 60.4 mg/l for northern pike (USEPA, 1980). Hardness does not appear to affect chronic toxicity. The acute-chronic ratios for different species range from 3 to 156 (USEPA, 1980). The more sensitive species tend to have lower ratios than the less sensitive species. In addition, the ratio seems to increase with hardness. Acute toxicity values for saltwater organisms range from 17 mg/l for a calanoid copepod to 600 mg/l for the shore crab (USEPA, 1980). A chronic value of 54 mg/l and an acute-chronic ratio of 3.4 is reported for the mysid shrimp (USEPA, 1980). Long-term exposure to 5 mg/l is fatal to the bay scallop.

Plants

Information was not found in the literature reviewed regarding toxicity of copper to plants.

Invertebrates

Information was not found in the literature reviewed regarding toxicity of copper to invertebrates.

Birds

Information was not found in the literature reviewed regarding toxicity of copper to birds.

Mammals

Sheep are very susceptible to acute or chronic copper toxicosis (Bostwick, 1982). Acute poisoning is caused by direct action of copper salts on the gastrointestinal tract, resulting in gastroenteritis, shock, and death. The toxic dose is about 200 mg/kg and is usually obtained through an accidental overdose of antihelminthic. Chronic ingestion of copper over a long period of time results in absorption and accumulation of copper by the liver. This type of chronic cumulative poisoning may suddenly develop into an acute hemolytic crisis. Copper

intake of 1.5 g/day for 30 days is known to be fatal for many breeds of sheep. Excessive copper may be stored in the liver as a result of excess copper ingestion, as a consequence of impaired liver function or in connection with a deficiency or excess of other trace elements. Sheep eliminate accumulated copper very slowly following cessation of exposure.

Swine may develop copper poisoning at levels of 250 mg/kg in the diet unless zinc and iron levels are increased. Toxicosis develops with hypochromic microcytic anemia, jaundice, and marked increases in liver and serum copper levels are well as serum aspartate amino transferase levels. High copper levels may be found in swine because of the practice of feeding them high copper diets to increase daily weight gain. However, swine rapidly eliminate copper once it is removed from the diet. Cattle are more resistant to copper in the diet than sheep or swine. Copper toxicity in ruminants can be counteracted by including molybdenum and sulfate in the diet.

REGULATIONS AND RECOMMENDED GUIDELINES

Ambient Water Quality Criteria (USEPA, 1986):

Aquatic Life (Freshwater)

Acute toxicity: $e^{[0.905\ (\ln\ (\text{hardness})) - 1.413]}$ mg/liter
At hardness of 50, 100, or 200 mg/l $CaCO_3$ the acute criteria are 9.2, 18 and 34 mg/liter.

Chronic toxicity: $e^{[0.905\ (\ln\ (\text{hardness})) - 1.785]}$ mg/liter
At hardness of 50, 100, or 200 mg/l $CaCO_3$ the chronic criteria are 6.5, 12 and 21 mg/liter.

Aquatic Life (Saltwater)

Acute toxicity: 2.9 mg/liter
Chronic toxicity: 2.9 mg/liter

Human Health

Organoleptic criterion: 1 mg/liter

National Primary Drinking Water Standards (USEPA):

1.3 mg/liter (Proposed MCLG 53 Federal Register 31516, Thursday, August 18, 1988)

OSHA PEL 29 CFR 1910.100

TWA = 1.0 mg/m^3 (dust and mist)

TWA = 0.1 mg/m^3 (fume)

ACGIH TLV

TWA = 1.0 mg/m^3 (dust and mist)

TWA = 0.2 mg/m^3 (fume)

DOSE-RESPONSE ASSESSMENT

The human dose-response parameter estimates for carcinogens and noncarcinogens are computed differently by USEPA; therefore, these estimates are presented separately below.

Carcinogenic Effects

USEPA has not developed oral or inhalation cancer potency estimates for copper.

Noncarcinogenic Effects

The oral intake reference dose (RfD) for copper is 0.0757 mg/kg/day. The RfD is based on a study addressing acute human clinical case studies (Chuttani, 1985) in which 5.3 mg copper was the lowest oral dose at which gastrointestinal effects were observed. The inhalation intake RfD has not been currently derived by the USEPA.

Oral RfD = 0.0757 mg/kg/day

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DDT/DDD/DDE

SUMMARY

DDT is an organochlorine pesticide, which together with its metabolites is very persistent in the environment. DDT, DDE, and DDD have been shown to be carcinogenic in mice, causing liver tumors but also increasing the incidence of lung tumors and lymphomas. Chronic exposure can damage the central nervous system and liver. In addition, DDT is a reproductive toxin. DDT and other organochlorine pesticides are highly toxic to aquatic organisms and readily bioconcentrate in their tissues. Bioaccumulation and subsequent biomagnification processes are responsible for the decreased reproductive success of many bird species.

Technical DDT is a mixture containing 65-80 percent p,p′-DDT, 15-20 percent o,p′-DDT, up to 4 percent p,p′-DDD, and traces of other materials. Metabolites of DDT include p,p′-DDE and o,p′-DDD. The DDT isomers and metabolites are usually found together and generally have similar properties; therefore, they are considered together. Where differences occur, the specific isomer is identified. DDT is used to refer to the combination of technical material and metabolites. Specific DDT isomers are identified as such.

CAS Number:
- p,p′-DDT: 50-29-3
- o,p′-DDT: 789-02-6
- p,p′-DDD: 72-54-8
- o,p′-DDD: 53-19-0
- p,p′-DDE: 72-55-9

Chemical Formula:
- p,p′- and o,p′-DDT: $C_{14}H_9Cl_5$
- p,p′- and o,p′-DDD: $C_{13}H_{10}Cl_4$
- p,p′- and o,p′-DDE: $C_{14}H_8Cl_4$

IUPAC Name:
- p,p′-DDT: 1,1,1-Trichloro-2,2-bis(4-chlorophenyl) ethane
- o,p′-DDT: 1,1,1-Trichloro-2-(2-chlorophenyl)-2-(4-chlorophenyl)ethane
- p,p′-DDD: 1,1-Dichloro-2,2-bis(4-chlorophenyl)ethane
- o,p′-DDD: 1,1-Dichloro-2,2-bis(4-chlorophenyl)ethane

Important Synonyms and Trade Names:

DDT: Dichlorodiphenyltrichloroethane, dicophane, chlorophenotane, Gesarol, Neocid
p,p'-DDD: TDE, Rothane

CHEMICAL AND PHYSICAL PROPERTIES

Molecular Weight: o,p'- and p,p'- DDT: 354.5
DDD: 320
DDE: 318

Boiling Point: DDT: 260°C

Melting Point: DDT: 109°C
DDD: 112°C
DDE: 90°C
88.4°C (Burrows et al., 1979)

Solubility in Water: p,p'-DDT: 5.5 mg/liter
o,p'-DDT: 26 mg/liter
p,p'-DDD: 20 mg/liter
DDE: 14 mg/liter

Solubility in Organics: DDT: Soluble in acetone, benzene, cyclohexanane, morpholine, pyridine, and dioxane

Log Octanol/Water Partition Coefficient (Kow):

DDT:	3.98-6.19	(Cited in Hansch and Leo, 1979)
	5.98	(Kenaga, 1980)
	6.19	(Rao and Davidson, 1983)
	6.36	(Davies and Dobbs, 1984)
	5.98	(Lyman et al., 1982)
	5.98; 6.19; 6.28; 6.36	(Geyer et al., 1984)
	4.0-7.48	(Kadeg et al., 1986 Range and geometric mean of 20 literature values) (geometric mean = 6.07)

p,p'-DDT: 3.98
p,p'-DDD: 5.99
o,p'-DDD: 6.08

DDE:	5.69	(Rao and Davidson, 1983)
	5.60	(Kadeg et al., 1986)
	7.00	(USEPA, 1986a)

Soil/Water Partition coefficient (Koc):

p,p'-DDE:

50,100	(Sabljic, 1984) (experimental)
147,900	(Kadeg et al., 1986) literature value
19,350; 662,200	(Kadeg et al., 1986) (log Kow = 4.86, 7.0)
10,490; 153,100	(Lyman et al., 1982) Eqn 4-8 (log Kow = 4.86, 7.0)
17,620; 818,500	(Lyman and Loreti, 1987) (log Kow = 4.86; 7.0)
4,400,000	(USEPA, 1986a)

p,p'-DDT:

23,800	(Kenaga, 1980) Table III (experimental)
140,000	(Chiou et al., 1979) (experimental)
243,000	(Rao and Davidson, 1983) Table I
4×10^6-43,650	(Kadeg et al., 1986) (Range and geometric mean of 17 literature values) (geometric mean = 302,000)

Bioconcentration Factor:

p,p'-DDE:

13,900	(Lyman et al., 1982) Eqn 5-2 (log Kow = 7.07)
12,430	(Lyman et al., 1982) Eqn 5-2 (log Kow = 5.69)
2,043	(Davies and Dobbs, 1984) Eqn A (S = 0.12)
25,362	(Davies and Dobbs, 1984) Eqn B (log Kow = 7.07)
980	(Davies and Dobbs, 1984) Eqn C (log Kow = 5.60)
3,400	(Davies and Dobbs, 1984) Eqn B (log Kow = 5.60)
10,600	(Lyman et al., 1982) Eqn 5-2 (log Kow = 5.60)
100,000	(Davies and Dobbs, 1984) Table 2 (experimental)
366 - 9,659	(Davies and Dobbs, 1984) Eqn B (log Kow = 3.98 - 6.36)

p,p'-DDT:

61,600; 84,500	(Kenaga, 1980) Table 3 (experimental)
623 - 29,800	(Lyman et al., 1982) Eqn 5-2 (log Kow = 3.98 - 6.19)
20,600	(Lyman et al., 1982) Eqn 5-2 (log Kow = 5.98)
40,100	(Lyman et al., 1982) Eqn 5-2 (log Kow = 6.36)
27,436 - 13,913	(Davies and Dobbs, 1984) Eqn A (S = 0.0012 - 0.004)
1,710	(Davies and Dobbs, 1984) Eqn C (log Kow = 6.07)
6,483	(Davies and Dobbs, 1984) Eqn B (log Kow = 6.07)
24,200	(Lyman et al., 1982) Eqn 5-2 (log Kow = 6.07)

Vapor Pressure:

p,p'-DDT:	1.9×10^{-7} mm Hg at 25°C
p,p'-DDT:	7.3×10^{-7} mm Hg at 30°C
o,p'-DDT:	5.5×10^{-6} mm Hg at 30°C
p,p'-DDD:	1.0×10^{-6} mm Hg at 30°C
o,p'-DDD:	1.9×10^{-6} mm Hg at 30°C
DDE:	6.5×10^{-6} mm Hg at 20°C (USEPA, 1979)

Henry's Law Constant:

DDD:	7.96×10^{-6} atm-m^3/mole (USEPA, 1985a)
DDE:	1.1×10^{-4} atm-m^3/mole (calculated)
	6.8×10^{-5} atm-m^3/mole (USEPA, 1985a)
	2.86×10^{-3} Dimensionless
DDT:	9×10^{-5} atm-m^3/mole (calculated)
	5.13×10^{-4} atm-m^3/mole (USEPA, 1985a)
	2.16×10^{-2} Dimensionless

TRANSPORT AND FATE

DDT and its metabolites are very persistent in the environment. Volatilization is not likely to be an important transport process from soil and water for DDT and its metabolites as evidenced by their low vapor pressures. The half-life of DDT in the atmosphere is not certain; however it is lost from the atmosphere by rain and photochemical degradation (USEPA, 1984).

The range of the Kocs is reported above indicates that sorption of DDT and its metabolites to soils/sediments and dissolved organic material will occur. Pavlou (1980) estimates that sorption of chlorinated hydrocarbon pesticides is very high. The combined low water solubility and high organic partitioning suggest that DDT will exhibit little environmental mobility. The half-life of DDT in soil is estimated to range between 3 and 15 years (USEPA, 1984).

Although it occurs slowly, p,p'-DDT, o,p'-DDT, and DDD are ultimately biotransformed in the environment (microorganisms) to form bis(2-chlorophenyl) methanone. In aquatic environments, indirect photolysis may also be important for p,p'DDT and o,p'-DDT. For DDE, direct photolysis is a more important fate process in the environment, although biotransformation may also be important.

A range of experimental and estimated BCFs for DDT and its metabolites in fish is reported above. Biomagnification of DDT and its metabolites has been demonstrated in many species, most notably in raptors. ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors indicates that significant bioconcentration and biomagnification of DDT residues can occur.

HEALTH EFFECTS

DDT, DDE, and DDD have been shown to be carcinogenic in mice, causing liver tumors, lung tumors, and lymphomas. DDT and its isomers have been classified according to USEPA's Proposed Guidelines for Carcinogen Risk Assessment in USEPA's Group B2 (probable human carcinogen), based on inadequate evidence for carcinogenicity in humans and sufficient evidence in animals (USEPA, 1984). DDT does not appear to be mutagenic, but it has caused chromosomal damage in some studies. The National Toxicology Program (1986) reports positive results for mutagenic activity with DDE utilizing mouse lymphoma cells in recently completed tests. There is no evidence that DDT is a teratogen. However, it is a reproductive toxin, causing reduced fertility, reduced growth of offspring, and fetal toxicity in rats (NIOSH, 1982).

Chronic exposure to DDT leads to a number of adverse effects of the liver and central nervous system (CNS). DDT induces various microsomal enzymes and therefore may affect the metabolism of steroid hormones and exogenous chemicals. Other effects on the liver include hypertrophy of the parenchymal cells and increased fat deposition. In the CNS, exposure to DDT causes behavioral effects such as decreased aggression and decreased conditional reflexes. Acute exposure to large doses or chronic exposure to lower doses causes seizures. The oral LD_{50} is between 113 and 450 mg/kg for the rat and is generally higher for other animals.

TOXICITY TO WILDLIFE AND DOMESTIC ANIMALS

Aquatic Life

DDT has been extensively studied in freshwater invertebrates and fishes and is quite toxic to most species. The range of toxicities to these organisms was 0.18 to 1,800 mg/l and the freshwater final acute value for DDT and it's isomers were determined by USEPA to be 1.1 mg/l (USEPA, 1980). Only one chronic toxicity test on aquatic species was reported. This test indicated that the acute chronic ratio for DDT may be high (65 in the reported study), but the data were insufficient to allow calculation of a final acute-chronic ratio.

Plants

Information was not found in the literature reviewed regarding toxicity of DDT, DDD, and DDE to plants.

Invertebrates

Information was not found in the literature reviewed regarding toxicity of DDT, DDD, and DDE to invertebrates.

Birds

DDT, DDD, DDE and other persistent orgnochlorine pesticides are primarily responsible for decreases in the reproductive capabilities and consequently in the populations of some fish-eating birds particularly the bald eagle, brown pelican, and osprey. DDT has also been shown to significantly decrease populations of other species of waterbirds, raptors, and passerines (EOP, 1971).

Mammals

Information was not found in the literature reviewed regarding toxicity of DDT, DDD, and DDE to mammals.

REGULATIONS AND RECOMMENDED GUIDELINES

Ambient Water Quality Criteria (USEPA, 1986b):

Aquatic Life (Freshwater)

DDT:

Acute toxicity: 1.1 mg/liter
Chronic toxicity: 0.001 mg/liter

Aquatic Life (Saltwater)

Acute toxicity: 0.13 mg/liter
Chronic toxicity: 0.001 mg/liter

DDD and DDE: The available data are not adequate for establishing Ambient Water Quality Criteria. However, USEPA does report the lowest values known to be toxic in aquatic organisms.

Aquatic Life (Freshwater)

Acute toxicity: DDD: 0.06 mg/liter
DDE: 1050 mg/liter

Chronic toxicity: DDD and DDE: No data available in the literature review.

Aquatic Life (Saltwater)

Acute toxicity: DDD: 3.6 mg/liter
DDE: 14 mg/liter

Chronic toxicity: DDD and DDE: No data available in the literature review.

Human Health

Due to the carcinogenicity of DDT and its isomers the ambient water criterion is set at zero. However, estimates of the carcinogenic risks associated with lifetime exposure from ingestion of contaminated water and contaminated aquatic organisms are:

Risk	Concentration
10^{-4}	2.4 ng/liter
10^{-5}	0.24 ng/liter
10^{-6}	0.024 ng/liter
10^{-7}	0.0024 ng/liter

OSHA PEL 29 CFR 1910.1000

TWA = 1 mg/m^3

ACGIH TLV

TWA = 1 mg/m^3

DOSE-RESPONSE ASSESSMENT

The human dose-response parameter estimates for carcinogens and noncarcinogens are computed differently by USEPA; therefore, these estimates are presented separately below.

Carcinogenic Effects

The Cancer Assessment Group (CAG) of USEPA has derived an oral cancer potency estimate for DDT of 0.34 $(mg/kg/day)^{-1}$ (USEPA, 1990).

The CAG has also derived an inhalation cancer potency estimate of 0.34 $(mg/kg/day)^{-1}$ (USEPA, 1990).

Oral Cancer Potency Estimate: 0.34 $(mg/kg/day)^{-1}$ (USEPA, 1990).
Inhalation Cancer Potency Estimate: 0.34 $(mg/kg/day)^{-1}$ (USEPA, 1990).

Noncarcinogenic Effects

The USEPA has not currently derived a reference dose (RfD) for DDT, DDD, DDE.

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DIBROMOCHLOROPROPANE

SUMMARY

Dibromochloropropane (DBCP) is a persistent and environmentally mobile pesticide. Formerly, DBCP was used as a soil fumigant and nematocide. It is carcinogenic in mice and rats and mutagenic in bacterial systems and mammalian cell cultures. It causes mammary tumors (female rats) and forestomach tumors when administered orally. When administered via inhalation, it causes nasal, tongue, and lung tumors. Men occupationally exposed to DBCP exhibit abnormally low sperm counts. Animal studies have shown that DBCP has adverse effects on the testes, liver, kidneys, respiratory tract, central nervous system, and blood cells.

CAS Number: 96-12-8

Chemical Formula: $C_3H_5Br_2Cl$

IUPAC Name: 1,2-Dibromo-3-chloropropane

Important Synonyms and Trade Names: DBCP, Fumazone, Nemagon

CHEMICAL AND PHYSICAL PROPERTIES

Molecular Weight: 236.36

Boiling Point: 196°C (Berkowitz et al., 1978)

Melting Point: 6°C
5°C (Berkowitz et al., 1978)

Specific Gravity: 2.093 at 14°C

Solubility in Water: 1230 mg/liter (USEPA, 1985a)

Solubility in Organics: Miscible with oils, dichloropropane, and isopropyl alcohol

Log Octanol/Water Partition Coefficient (Kow):

2.29 (Lyman et al., 1982) Fragment Method
2.43 (USEPA, 1985a)

Soil/Water Partition Coefficient (Koc):

130 (Sabljic, 1984) Table I (experimental)
175 (Lyman and Loreti, 1987) (log Kow = 2.29)
225 (Lyman and Loreti, 1987) (log Kow = 2.43)

Bioconcentration Factor:

41.4 (Lyman et al., 1982) Eqn 5-2 (log Kow = 2.43)
67.5 (Lyman et al., 1982) Eqn 5-2 (log Kow = 2.71)
11.2 (Davies and Dobbs, 1984) Eqn A (S = 1,230)
63 (Davies and Dobbs, 1984) Eqn B (log Kow = 2.71)
43.5 (Davies and Dobbs, 1984) Eqn B (log Kow = 2.43)
35.9 (Davies and Dobbs, 1984) Eqn B (log Kow = 2.29)
19.8 (Davies and Dobbs, 1984) Eqn C (log Kow = 2.29)
27.6 (Davies and Dobbs, 1984) Eqn C (log Kow = 2.43)
32.4 (Lyman et al., 1982) Eqn 5-2 (log Kow = 2.29)

Vapor Pressure: 0.8 mm Hg at 21°C (USEPA, 1985a)
1.1 mm Hg at 25°C (estimated; Lyman et al., 1982)

Henry's Law Constant: 3.5×10^{-4} atm-m^3/mole at 20°C (Burlinson et al., 1982)
1.47×10^{-2} Dimensionless
3.11×10^{-4} atm-m^3/mole (USEPA, 1985b)
1.31×10^{-2} Dimensionless

TRANSPORT AND FATE

Dibromochloropropane (DBCP) is a persistent pesticide. The major route of its removal from soil and aqueous systems is by volatilization. DBCP is decomposed slowly in soil both by microbial action and by hydrolysis (USEPA, 1985b). DBCP may be converted to n-propanol, bromide, and chloride by soil/water culture (Berkowitz et al., 1978). A range of estimated and experimental Kocs is reported above and indicates that sorption of DBCP to soils/sediments and dissolved organic material will occur. The combined water solubility and organic partitioning data for DBCP suggest that this compound will exhibit some degree of environmental mobility.

Plant uptake can occur with DBCP levels generally highest in the root portion. Bromide ion has also been shown to be present in increased levels in plants grown in DBCP-treated fields (Guinn and Potter, 1962), and may be due to microbial or plant enzyme activity.

A range of estimated BCFs for DBCP is also reported above. ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains.

The magnitude of the concentration factors suggests that appreciable bioconcentration or biomagnification of DBCP residues is not likely to occur.

HEALTH EFFECTS

DBCP was found to be carcinogenic in several animal bioassays via oral, inhalation and dermal routes of exposure and mutagenic in the Ames assay system. In studies with DBCP, the NTP reported no effects on dominant lethal frequency in mice receiving intraperitoneal and subcutaneous injections (NTP, 1985). It has also caused somatic cell mutations and chromosomal aberrations in Drosophila melanogaster (USEPA, 1985b). Chromosome aberrations and positive evidence of sister chromatid exchange have been reported in Chinese hamster ovary cells (NTP, 1986). In a gavage study using mice and rats, DBCP produced significantly increased incidences of squamous-cell carcinomas of the forestomach in both sexes of both species and mammary adenocarcinomas in female rats (USEPA, 1985b). In an inhalation study, rats had increased incidence of nasal cavity tumors and tumors of the tongue, while mice had increased incidence of nasal cavity tumors and lung tumors (USEPA, 1985b). Dermal exposure resulted in increased incidence of skin and lung tumors in mice (USEPA, 1985b). DBCP has been classified according to USEPA's Proposed Guidelines for Carcinogenic Risk Assessment in Group B2 (probable human carcinogen) based on positive results in animal studies and inadequate data in humans (50 Federal Register 46989, Wednesday, November 13, 1985).

Men occupationally exposed to DBCP during its manufacture were found to have abnormally low sperm counts (USEPA, 1985b). Male rats exposed to DBCP during subchronic toxicity studies were also found to have abnormally low sperm cells as well as degenerative changes in the seminiferous tubules, decreased weight of the testes, and an increased proportion of abnormal sperm cells (USEPA, 1985b). Liver and kidney effects have also been noted in animal studies. Effects range from dilatation of the sinusoids and centrilobular congestion to cirrhosis and necrosis in the liver. Cloudy swelling of the epithelium of the proximal convoluted tubules and increased amounts of interstitial tissue have been found in the kidneys (USEPA, 1985b). Effects on blood cells were also noted in several studies. These effects

include severe leukopenias and anemias in exposed monkeys and decreased activity of phagocytic cells in exposed rats (USEPA, 1985b).

TOXICITY TO WILDLIFE AND DOMESTIC ANIMALS

Aquatic Life

Exposure to a water concentration of 1 mg/l DBCP for 24 hours produced a 90 percent mortality in clam larvae.

Plants

Information was not found in the literature reviewed regarding toxicity of DBCP to plants.

Invertebrates

At a concentration of 20 gallons DBCP per acre, 100 percent of exposed earthworms died in 1 day. At a use rate of 5 pounds per acre, DBCP killed 87 percent of Lumbricus and 28 percent of the Helodrolus sp. in 32 days.

Birds

The acute oral LD_{50} value of DBCP to female mallard ducks is 66.8 mg/kg and 156 mg/kg in female pheasants. Both avian LD_{50} values are lower than the acute oral LD_{50} value of the rat indicating an increased sensitivity of these animals.

Mammals

The acute oral LD_{50} value of DBCP in rats is 400 mg/kg. Limited information was available in literature regarding the toxicity of DBCP to mammals.

REGULATIONS AND RECOMMENDED GUIDELINES

National Primary Drinking Water Standard (USEPA):

Zero (Proposed RMCL; 50 Federal Register 46988, Wednesday, November 13, 1985).

NIOSH REL: 10 ppb

OSHA PEL 29 CFR 1910.1044

TWA = 1 ppb

DOSE-RESPONSE ASSESSMENT

The human dose-response parameter estimates for carcinogens and noncarcinogens are computed differently by USEPA; therefore, these estimates are presented separately below.

Carcinogenic Effects

The Cancer Assessment Group (CAG) of USEPA has derived an oral cancer potency estimate for DBCP of 1.4 $(mg/kg/day)^{-1}$ (USEPA, 1985b).

The CAG has not derived an inhalation cancer potency estimate.

Oral Cancer Potency Estimate: 1.4 $(mg/kg/day)^{-1}$ (USEPA, 1985b).

Noncarcinogenic Effects

The USEPA has not currently derived a reference dose (RfD) for DBCP.

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DICYCLOPENTADIENE

SUMMARY

No data on the toxicity of dicyclopentadiene (DCPD) to humans was located in available literature. DCPD was not mutagenic in standard microbial assays (both activated and inactivated). No evidence of toxicity was observed following subchronic dietary administration to rats, mice, or dogs at levels ranging up to 750, 273, or 1,000 ppm, respectively. No reproductive effects occurred following DCPD exposure in male and female rats, nor were doses of DCPD teratogenic when administered to pregnant rats during gestation days 6-15.

CAS Number: 77-73-6

Chemical Formula: $C_{10}H_{12}$

IUPAC Name: Dicyclopentadiene

Important Synonyms and Trade Names: DCPD

CHEMICAL AND PHYSICAL PROPERTIES

Molecular Weight: 132

Melting Point: 32.9°C (Rosenblatt et al., 1975)

Boiling Point: 170°C (Cogley and Foy, 1978)

Solubility in Water: 20 mg/l (estimated; Lyman et al., 1982)

Log Octanol/Water Partition Coefficient (Kow): 3.14 (Lyman et al., 1982) Fragment Method

Soil to Water Partition Coefficient (Koc):

1,217 (Lyman et al., 1982) Eqn 4-8 (log Kow = 3.14)
806 (Lyman and Loreti, 1987) (log Kow = 3.14)

Bioconcentration Factor:

53 (Bentley et al., 1976) (experimental)
114 (Davies and Dobbs, 1984) Eqn A (S = 20)
143 (Lyman et al., 1982) Eqn 5-2 (log Kow = 3.14)
115 (Davies and Dobbs, 1984) Eqn B (log Kow = 3.14)
53.9 (Davies and Dobbs, 1984) Eqn C (log Kow = 3.14)

Specific Gravity: 0.98 (Rosenblatt et al., 1982)

Vapor Pressure: 2.2 mm Hg at 25°C (estimated; Rosenblatt et al., 1975)
1.4 mm Hg at 20°C (estimated; Rosenblatt et al., 1975)

Henry's Law Constant: 1.9×10^{-2} atm-m^3/mole (calculated)
8.28×10^{-1} Dimensionless
1.2×10^{-2} atm-m^3/mole (calculated)

TRANSPORT AND FATE

The vapor pressure of DCPD indicates that volatilization (evaporation) from surface water to the atmosphere can occur. The chemical fate of DCPD in the atmosphere is not definitively known; however, photodegradation may occur. DCPD is virtually insoluble in water (Cogley and Foy, 1978). A range of estimated Kocs is reported above and indicates that sorption of DCPD to soils/sediments and dissolved organic materials will occur. The combined low water solubility and high organic partitioning coefficients suggest that dicyclopentadiene will not be mobile in the environment. The half-life of DCPD in soil ranges from 6 months to 1 year depending on ambient conditions (Cogley and Foy, 1978). Degradation to more stable forms (degradation forms were not reported) occurs, and the reported half-lives of these products are greatly increased, ranging from one to greater than 5 years (Cogley and Foy, 1978). Spanggord et al. (1979) reported an estimated half-life of 4-7 years for DCPD incubated (25°C) soil samples.

Biodegradation in aquatic systems is not likely to be extensive (Spanggord et al., 1979). An estimated 76-day or greater half-life of DCPD in water samples was also reported by Spanggord et al. (1979), based upon sunlight exposure (photolysis) tests. A 5.3-day half-life for DCPD in water samples (25°C, without recharge) was also observed. Uptake of less than 100 ppm DCPD was observed in plants which were grown in hydroponic solutions (1,000 ppm) (O'Donovan and Woodward, 1977). Evidence of stunted growth was also seen in plants at this concentration.

A range of experimental and estimated BCFs for dicyclopentadiene is also reported above. ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately

100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors suggests that appreciable bioconcentration or biomagnification of DCPD residues is not likely to occur.

HEALTH EFFECTS

No data on the toxicity of DCPD in humans was located in available literature. DCPD was not mutagenic in a variety of microbial assays both with and without metabolic activation (Hart, 1980). Data on the carcinogenicity of DCPD was not found in the literature review. Dicyclopentadiene was minimally irritating to rabbit skin and did not produce evidence of systemic toxicity following application (Hart, 1976). No evidence of toxicity followed dietary administration for 90 days to rats at levels up to 750 ppm or to mice at levels up to 273 ppm (Hart, 1976). Hart (1980) administered DCPD to beagle dogs in their diets for 13 weeks at concentrations of 100, 300, or 1,000 ppm. Clinical pathological evaluations, including analyses of clinical chemical constituents of serum, urine, and hemograms, were performed at monthly intervals. Tissues from control and treated dogs were compared histopathologically. No significant toxicity was observed with the possible exception of minor indications of intestinal distress expressed as vomiting and soft stools among treated groups, especially the highest dose (Hart, 1980). The NOAEL identified from this study was 1,000 ppm (25 mg/kg/day). No effects on fertility indices, live-to-total pup ratios, mean litter sizes, pup survival indices or mean body weights of pups post partum were observed in rats given 80 or 750 ppm DCPD in the diet prior to mating. Likewise, no dose-related teratogenic effects were observed in pregnant females administered 80, 250, or 750 ppm in the diet during days 6-15 of gestation (Hart, 1980). DCPD had oral LD_{50}'s of 520 and 378 mg/kg in male and female rats and 190 and 250 mg/kg in male and female mice (Hart, 1976).

TOXICITY TO WILDLIFE AND DOMESTIC ANIMALS

Aquatic Life

The 90-hour LC_{50} for dicyclopentadiene for fathead minnows is 31.1 mg/l (Bentley et al., 1976).

Plants

Information was not found in the literature reviewed regarding toxicity of dicyclopentadiene to plants.

Invertebrates

Information was not found in the literature reviewed regarding toxicity of dicyclopentadiene to invertebrates.

Birds

Dicyclopentadiene was found to be relatively nontoxic to mallard ducks (Aulerich et al., 1979). An oral LD_{50} could not be determined, even when levels administered were as high as 40,000 mg/kg. The oral LD_{50} in Bobwhite quail was 1,010 mg/kg. The biological half-life of DCPD residues in ducks and quail fed ^{14}C-DCPD-treated diets averaged 12.7 hours and was not concentrated in adipose tissue of either species.

Mammals

The oral LD_{50} in mink is 1,00 mg/kg.

REGULATIONS AND RECOMMENDED GUIDELINES

OSHA PEL 29 CFR 1910.1000

TWA = 5 ppm

ACGIH TLV

TWA = 5 ppm

DOSE-RESPONSE ASSESSMENT

The human dose-response parameter estimates for carcinogens and noncarcinogens are computed differently by USEPA; therefore, these estimates are presented separately below.

Carcinogenic Effects

USEPA has not developed oral or inhalation cancer potency estimates for DCPD.

Noncarcinogenic Effects

The oral intake reference dose (RfD) for dicyclopentadiene is 0.03 mg/kg/day based on a study assessing the toxicity of dietary dicyclopentadiene (690 ppm, 32 mg/kg/day for males) in rats over three generations (USEPA, 1990). The RfD for inhalation intake is 0.61 mg/kg/day based on a subchronic inhalation study in which rats were exposed to 1 ppm DCPD 6 hours/day, 5 days/week for 90 days (Dodd et al., 1982).

Oral RfD = 0.03 mg/kg/day

Inhalation RfD = 0.61 mg/kg/day

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ENDRIN

SUMMARY

Endrin, an isomer of Dieldrin, is an insecticide belonging to the chemical class of cyclodienes. It is retained in soils and sediments and is very persistent in the environment by virtue of its structure and physical/chemical properties. It is readily bioaccumulated by aquatic organisms. Endrin is acutely toxic to mammals, aquatic organisms, and terrestrial wildlife. It has not yet been shown to be carcinogenic or mutagenic, but is a potent teratogen and reproductive toxin.

CAS Number: 72-20-8

Chemical Formula: $C_{12}H_8Cl_6O$

IUPAC Name: 1,2,3,4,10 Iexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a octahydro-endo-1,4:5,8-dimethanonaphthalene

Important Synonyms and Trade Names: Endrin, hexadrin, mendrin

CHEMICAL AND PHYSICAL PROPERTIES

Molecular Weight: 380.9

Melting Point: Decomposes at 235°C

Specific Gravity: 1.65 at 25°C

Solubility in Water: 0.25 mg/liter at 25°C
0.23 mg/liter at 25°C (Rosenblatt et al., 1975)
0.1 mg/liter (Rao and Davidson, 1983) Table 1
0.024 mg/liter (Kenaga, 1980) Table III

Solubility in Organics: Soluble in acetone, benzene, carbon tetrachloride, hexane, and xylene

Log Octanol/Water Partition Coefficient (Kow): 5.34 (Kenaga, 1980) Table III
3.21 (Rao and Davidson, 1983) Table II
4.44 (Kadeg et al., 1986)

Soil/Water Partition Coefficients (Koc):

1,312; 26,510	(Lyman et al., 1982) Eqn 4-8 (log Kow = 3.20; 5.60)
34,000	(Kenaga, 1980) Table III
897; 66,440	(Lyman and Loreti, 1987) (log Kow = 3.20; 5.60)
1,249; 5,640	(Kadeg et al., 1986) (log Kow = 3.20; 5.60)
3,630	(Kadeg et al., 1986), (geo. mean 2 literature values)

Bioconcentration Factor:

4,050	(Kenaga, 1980) Table 3 (experimental)
1,360	(Kenaga, 1980) Table 3 (experimental)
2,377	(Davies and Dobbs, 1984) Eqn B (log Kow = 5.34)
1,415.7	(Davies and Dobbs, 1984) Eqn A (S = 0.23)
5,012	Davies and Dobbs Table 2 (experimental)
6,736	(Lyman et al., 1982) Eqn 5-2 (log Kow = 5.34)
1,043	(Davies and Dobbs, 1984) Eqn C (log Kow = 5.34)
250	(Davies and Dobbs, 1984) Eqn C (log Kow = 4.44)
690	(Davies and Dobbs, 1984) Eqn B (log Kow = 4.44)
1,390	(Lyman et al., 1982) Eqn 5-2 (log Kow = 4.44)
1,640	(Argyle, 1973) (experimental)
13,000	(Hermanutz, 1978) (experimental)

Vapor Pressure: 2.7×10^{-7} mm Hg at 25°C (Rao and Davidson, 1983)
2.0×10^{-7} mm Hg at 25°C (Rosenblatt et al., 1975)

Henry's Law Constant: 4.4×10^{-7} atm-m^3/mole (calculated)
1.8×10^{-5} Dimensionless
4.2×10^{-6} atm-m^3/mole (calculated)
1.8×10^{-4} Dimensionless

TRANSPORT AND FATE

Endrin is quite persistent in the environment. Volatilization from soil surfaces and from surface water is not likely to be an important transport process (Nash, 1983) in light of its very low vapor pressure. For the small portion that may volatilize, photolysis to delta-keto endrin and endrin aldehyde are important chemical fate processes.

A range of estimated Kocs is reported above and indicates that sorption of endrin to soils/sediments and dissolved organic materials will occur. Pavlou (1980) estimates that sorption of organochlorine pesticides such as endrin is very high. The combined low water solubility and high organic partitioning of endrin indicates that little environmental mobility

will occur. Rosenblatt et al. (1975) report less than 10 cm of movement in situ following 150 cm of rainfall. Microbial degradation by soil microorganisms occurs but appears to be limited (Rosenblatt et al., 1975). The extent of utilization and the decomposition products were not reported. Endrin has a relatively long half-life in soil which may span upwards of 10 years.

Uptake in plants varies with species. For example, root crops (potatoes) grown in treated soil exhibited levels about twice that of the soil in which they were grown (Telekar et al., 1983). Levels in pasture crops appear to be less than those in soil (Chawla et al., 1981).

A range of experimental and estimated BCFs for endrin is also reported above. ASTM (1985) indicates that chemicals with BCFs less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors suggests that bioconcentration and potential biomagnification will occur.

HEALTH EFFECTS

Endrin has not been shown to be carcinogenic in several animal studies including the National Cancer Institute bioassay (50 Federal Register 47011, Wednesday, November 13, 1985). Endrin has not been shown to be mutagenic in microbial systems with or without activation (50 Federal Register 47011). Endrin has been classified according to USEPA's Carcinogenic Risk Assessment Guidelines in USEPA's Group D (not classifiable as to human carcinogenicity).

Endrin is a potent reproductive toxin and teratogen in experimental animals. Reproductive effects include fetal mortality and growth retardation, while teratogenic effects included cleft palate, open eye, clubbed foot, meningoencephales, and fused ribs. Chronic exposure to low levels of Endrin results primarily in nervous system damage; however, adverse effects to the heart, lungs, liver, and kidneys also occur. The acute toxicity of endrin is due to its effects on the central nervous system. The acute oral LD_{50} is 3 mg/kg in the rat and 1.37 mg/kg in mice (Sax, 1979).

TOXICITY TO WILDLIFE AND DOMESTIC ANIMALS

Aquatic Life

Endrin is very toxic to aquatic organisms. Freshwater fish were generally more sensitive than invertebrates, with species mean acute values ranging form 0.15 to 2.1 mg/l (USEPA, 1980). LC_{50} values for saltwater organisms ranged form 0.037 to 14.25 mg/l. Final acute values for freshwater and saltwater species were 0.18 mg/l and 0.037 mg/l, respectively (USEPA, 1980). An acute-chronic ratio of 4.0 was determined from chronic tests on freshwater and saltwater species. Therefore, the freshwater final chronic value was calculated to be 0.045 mg/l, and the saltwater final chronic value was determined to be 0.0093 mg/l (USEPA, 1980).

Plants

Information was not found in the literature reviewed regarding toxicity of endrin to plants.

Invertebrates

Information was not found in the literature reviewed regarding toxicity of endrin to invertebrates.

Birds

The LD_{50} values for a variety of birds are 5.64 mg/kg (mallard), 1.1 mg/kg (grouse), 1.2 mg/kg (quail), and 1.8 mg/kg (pheasant).

Mammals

Endrin is acutely toxic to terrestrial wildlife and domestic animals and has been used as a rodenticide and an avicide. It can also cause central nervous system effects and reproductive disorders following chronic exposure. Other effects observed in animals exposed to endrin include abnormal behavior, increased postnatal mortality, and increased fetal death. In terrestrial mammals, such as the mule deer and the domestic goat, the range of acute oral LD_{50}s are 6.25 - 12.5 mg/kg and 25-50 mg/kg, respectively (Hudson et al., 1984).

REGULATIONS AND RECOMMENDED GUIDELINES

Ambient Water Quality Criteria (USEPA, 1986):

Aquatic Life (Freshwater)

Acute toxicity: 0.18 mg/liter
Chronic toxicity: 0.0023 mg/liter

Aquatic Life (Saltwater)

Acute toxicity: 0.037 mg/liter
Chronic toxicity: 0.0023 mg/liter

Human Health

Criterion: 1.0 mg/liter (water and fish ingestion)

National Interim Primary Drinking Water Standard USEPA:

0.0002 mg/l (MCL; 40 CFR 141.12 Subpart B)

OSHA PEL 29 CFR 1910.1000

TWA = 0.1 mg/m^3 (skin)

ACGIH TLV

TWA = 0.1 mg/m^3 (skin)

DOSE-RESPONSE ASSESSMENT

The human dose-response parameter estimates for carcinogens and noncarcinogens are computed differently by USEPA; therefore, these estimates are presented separately below.

Carcinogenic Effects

USEPA has not classified endrin as a carcinogen, therefore cancer potency factors have not been developed.

Noncarcinogenic Effects

The oral intake reference dose (RfD) for endrin is 0.00025 mg/kg/day based on a study assessing the toxicity of dietary endrin (0.1, 0.5, 1.0, 2.0, and 4.0 ppm) in dogs over a period of 2 years. The inhalation intake RfD has not been determined for endrin at this time.

Oral RfD = 0.00025 mg/kg/day

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FLUOROACETIC ACID

SUMMARY

Fluoroacetic acid and its sodium salt, sodium fluoroacetate are acutely toxic to birds and mammals. Sodium fluoroacetate is used primarily as a rodenticide and is toxic as a result of its oxidative conversion to fluorocitrate in vivo. Fluorocitrate effectively blocks the tricarboxylic acid cycle which is an essential mechanism in mammals for energy production. Data on the environmental persistence of sodium fluoroacetate are lacking.

CAS Number: 144-49-0

Chemical Formula: CH_2FCOOH

IUPAC Name: 2-Fluoroacetic Acid

Important Synonyms and Trade Names: Fluoroethanoic Acid; Gifblaar Poison; MFA; Monofluoroacetic Acid

CHEMICAL AND PHYSICAL PROPERTIES

Molecular Weight: 78.04 (Merck, 1983)

Boiling Point: 165°C (Sax, 1979)

Melting Point: 33°C (Merck, 1983)

Specific Gravity: 1.369 (USEPA, 1985)

Solubility in Water: Soluble (Merck, 1983)

Solubility in Organics: Slightly soluble in petroleum ether

Log Octanol/Water Partition Coefficient (Kow): Data not found in literature review

Soil to Water Partition Coefficient (Koc): Data not found in literature review

Bioconcentration Factor: Data not found in literature review

Vapor Pressure: Data not found in literature review

Henry's Law Constant: Data not found in literature review

TRANSPORT AND FATE

Scant information is available on the transport and fate processes of fluoroacetic acid or its sodium salt, sodium fluoroacetate. Both are water soluble (USEPA, 1985; Gosselin, 1976). Sodium fluoroacetate is also nonvolatile (Gosselin, 1976) and therefore, losses from environmental media due to evaporation would not be expected to occur. Under normal conditions of pH in soil and water it is likely that the compound will be present as a salt rather than as a free acid. Potassium fluoroacetate is a natural toxic constituent of the South African plant Dichaepetalum cymosum (Peters et al., 1981). Fluoroacetate is also a natural constituent of some poisonous plants, notably Acacia georginae, a perennial shrub found in Australia (Gosselin, 1976). Neither plant is likely to occur naturally in the United States.

No data on the stability of fluoroacetic acid (or its sodium salt, sodium fluoroacetate) in air, soil, water or its potential for bioaccumulation were located in available literature. However, given the soluble nature of fluoroacetic acid and its sodium salt, bioconcentration would not be expected to occur.

HEALTH EFFECTS

Data presented are for sodium fluoroacetate, the salt of fluoroacetic acid. The fluoroacetate ion itself is not toxic, but is converted in vivo to fluorocitric acid (fluorocitrate), a potent inhibitor of the tricarboxylic acid cycle--an essential mechanism in energy production in mammalian cells (Gosselin, 1976). The block is a result of the inhibition of aconitase that regulates the conversion of citrate to isocitrate. The result is an accumulation of large quantities of citrate in the tissues (Casarett and Doull, 1980). Because the metabolic lesion involves an inhibition of oxidative energy metabolism, the heart and the central nervous system are the critical areas affected (Casarett and Doull, 1980). Symptoms of poisonings include nausea, vomiting, cardiac irregularities, cyanosis, and convulsions. Death is usually the result of ventricular fibrillation or respiratory failure. The estimated lethal dose for humans ranges from 2 to 10 mg/kg (Casarett and Doull, 1980).

Species differences are reported in the types of symptoms which precede death. Dogs usually die of convulsions or respiratory failure; however, in man, monkeys, horses, and rabbits,

central nervous system effects are often incidental with the principal complication arising from ventricular fibrillation (Casarett and Doull, 1980).

Few data are available on the effects of chronic poisoning with sodium fluoroacetate; however, renal changes similar to nephrosis have occurred in rats administered acutely lethal or repeated sublethal injections of fluorocitrate (Gosselin, 1976). In one reported case of chronic poisoning, a rabbit exterminator exposed repeatedly over a period of 10 years exhibited severe and progressive lesions of the renal tubular epithelium and milder hepatic neurologic and thyroid dysfunctions (Gosselin, 1976).

Data on reproductive effects, teratogenicity, carcinogenicity, or mutagenicity of fluoroacetic acid or related compounds was not located in available literature. The oral LD_{50} values in rats, mice and guinea pigs are 4.7, 7, and 0.47 mg/kg, respectively (NIOSH, 1983).

TOXICITY TO WILDLIFE AND DOMESTIC ANIMALS

Aquatic Life

Some poikilothermic animals reported to be resistant to fluoroacetate; notably, the South African clawed toad (Xenopus laevis) and some fish such as bass and the bream (Bauermeister et al., 1977). The intraperitoneal LD_{50} of fluoroacetate in rainbow trout (Salmo gairdneri) is 500 µmole/kg (Bauermeister et al., 1977). Acute oral toxicity (LD_{50}) for bullfrogs was 54.4 mg/kg (Hudson et al., 1984).

Plants

Information was not found in the literature reviewed regarding toxicity of fluoroacetic acid to plants.

Invertebrates

Information was not found in the literature reviewed regarding toxicity of fluoroacetic acid to invertebrates.

Birds

Acute oral toxicities, LD_{50}, are presented below for a variety of species (Hudson et al., 1984).

Species	LD_{50} (mg/kg)
Mallard ducks	9.11
Golden eagles	3.54
California quail	4.63
Japanese quail	12.8
Ring-necked pheasant	6.46
Chuker	3.51
Turkeys	4.76
Domestic pigeons	4.24
House sparrows	3.0

Mammals

Acute oral toxicity (LD_{50}) for domestic ferrets was 1.41 mg/kg and for mule deer was 0.33-1.0 mg/kg (Hudson et al., 1984). Hudson et al. (1984) reported secondary toxicity in tested ferrets fed live or dead mice previously dosed with 1, 2, 4, or 8 mg/kg sodium fluoroacetate. Only one ferret survived following the ingestion of one of two low-dose (2 mg/kg) mice.

REGULATIONS AND RECOMMENDED GUIDELINES

OSHA PEL 29 CFR 1910.1000

TWA = 0.05 mg/m^3 (sodium salt)

ACGIH TLV

TWA = 0.05 mg/m^3 (sodium salt)

STEL = 0.15 mg/m^3 (sodium salt)

DOSE-RESPONSE ASSESSMENT

The human dose-response parameter estimates for carcinogens and noncarcinogens are computed differently by USEPA; therefore, these estimates are presented separately below.

Carcinogenic Effects

USEPA has not developed oral or inhalation cancer potency estimates for fluoroacetic acid or sodium fluoroacetate.

Noncarcinogenic Effects

The USEPA has not currently derived a reference dose (RfD) for fluoroacetic acid.

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HEXACHLOROCYCLOPENTADIENE

SUMMARY

Hexachlorocyclopentadiene (HCPD) has not presently been shown to be carcinogenic in animals or humans; however, The National Cancer Institute has selected HCPD for testing. No evidence of mutagenicity has been established for HCPD in either mammalian or bacterial test systems. In animal studies, HCPD given orally resulted in toxic nephrosis in female mice and in male and female rats. Rats exposed to high concentrations of HCPD via inhalation experienced mortality, depressed body weights, increased kidney weights (females only), and pulmonary degenerative changes. HCPD has not resulted in teratogenic or embryotoxic effects following its administration to rabbits and rats; however, maternal toxicity was observed in treated rabbits.

CAS Number: 77-47-4

Chemical Formula: C_5Cl_6

IUPAC Name: 1,2,3,4,5,5-Hexachloro-1,3-cyclopentadiene

Important Synonyms and Trade Names: HCPD; Perchlorocyclopentadiene

CHEMICAL AND PHYSICAL PROPERTIES

Molecular Weight: 273

Melting Point: -9.6°C (USEPA, 1984)

Boiling Point: 239°C at 753 mm Hg (Hawley, 1977; Stevens, 1979)
234°C (Irish, 1963)

Specific Gravity: 1.715 at 15.5°C (Hawley, 1977)

Solubility in Organic Solvents: Miscible in hexane (Bell et al., 1978)

Solubility in Water: 2.1 mg/liter at 25°C (Dal Monte and Yu, 1977)
1.8 mg/liter at 28°C (Wolfe et al., 1982)
0.805 mg/liter at 25°C (Lu et al., 1975)
2.1 mg/liter at 25°C (USEPA, 1986a)

Log Octanol/Water Partition Coefficient (Kow): 3.52 (Lyman et al., 1982) Fragment Method
5.04 (Wolfe et al., 1982)

Soil/Water Partition Coefficients (Koc):

13,140 (Lyman et al., 1982) Eqn 4-8 (log Kow = 5.04)
24,330 (Lyman and Loreti, 1987) (log Kow = 5.04)
4,800 (USEPA, 1986a)

Bioconcentration Factor:

29 (Veith et al., 1979) (experimental)
11 (USEPA, 1980)
279 (Lyman et al., 1982) Eqn 5-2 (log Kow = 3.52)
195 (Davies and Dobbs, 1984) Eqn B (log Kow = 3.52)
107.6 (Davies and Dobbs, 1984) Eqn C (log Kow = 3.52)
179 (Davies and Dobbs, 1984) Eqn A (S = 9)
717 (Davies and Dobbs, 1984) Eqn C (log Kow = 5.04)
1,570 (Davies and Dobbs, 1984) Eqn B (log Kow = 5.04)
3,980 (Lyman et al., 1982) Eqn 5-2 (log Kow = 5.04)

Vapor Pressure:0.08 mm Hg at 25°C (Irish, 1963)
0.975 mm Hg at 62°C (Stevens, 1979)

Henry's Law Constant: 0.0137 atm-m^3/mole (USEPA, 1986a)
5.76×10^{-1} Dimensionless
0.027 atm-m^3/mole (Atallah et al., 1980; Wolfe et al., 1982)
1.13 Dimensionless

TRANSPORT AND FATE

HCPD is known to volatilize rapidly from water (USEPA, 1984); however, it is not likely to persist following its release to air. The estimated tropospheric residence time (Cupitt, 1980) is approximately 5 hours based on reactions with hydroxyl radicals and ozone (USEPA, 1984). Atmospheric photolysis of HCPD is likely since HCPD has a chromophore which absorbs light in the solar spectrum. The degradation products are thought to be ClCO, diacylchlorides, ketone, and free Cl radical (USEPA, 1984).

HCPD is known to photolyze in aqueous media. In flowing bodies of water, photolysis, hydrolysis, volatilization, and biodegradation will all contribute to the loss of HCPD. The photolytic half-life of HCPD in shallow water (<5 cm depth) is estimated to be 10 minutes (USEPA, 1984). Hydrolysis is much slower with a half-life ranging from 3-11 days at pHs of 5-9 and temperatures between 25 and 30°C (Wolfe et al., 1982).

The fate and transport of HCPD in soils is affected by its strong tendency to adsorb onto organic matter (USEPA, 1984). A range of estimated Kocs is reported above and indicates that sorption of HCPD to soils/sediments and dissolved organic material will occur. The combined low water solubility and high organic partitioning for HCPD suggests that this compound will not be an environmentally mobile contaminant. HCPD is known to be metabolized by a number of soil microorganisms (USEPA, 1984).

A range of estimated and experimental BCFs for HCPD is also reported above. American Society of Testing and Materials (ASTM, 1985) indicates that chemicals with BCFs less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the estimated concentration factors suggests that appreciable bioconcentration or biomagnification of HCPD residues would occur; however, experimental data appear to indicate that uptake is not considerable (USEPA, 1984).

HEALTH EFFECTS

Little data are available on the health effects of HCPD exposures in humans. The compound is very irritating to the eyes and mucous membranes and induces lacrimation, sneezing, and salivation. Repeated contact with the skin causes blistering burns, and inhalation causes pulmonary edema (USEPA, 1984).

Subchronic (90-day) oral exposures of mice to doses of HCPD (19, 38, 75, 150, 300 mg/kg) 5 days/week resulted in lesions of the forestomach in both sexes at 38 mg/kg (USEPA, 1984). At the highest dose all male mice died by day 8 and 3 females by day 14. In female mice the liver was enlarged, and toxic nephrosis was evident at doses greater than 75 mg/kg. In another phase of the study, rats were orally exposed to doses of 10, 19, 38, 75, and 150 mg/kg HCPD. Mortality and toxic nephrosis was observed in both males and females at doses >38 mg/kg (USEPA, 1984). Female rats exposed to 19 mg/kg exhibited lesions of the forestomach. A dose-related depression in bodyweight gain was also observed relative to controls. The acute oral LC_{50} of HCPD in rats ranges from 500-630 mg/kg (USEPA, 1980).

Rats and monkeys exposed subchronically (14 weeks) to HCPD via inhalation at doses of 0, 0.1, 0.05, and 0.20 ppm exhibited no treatment-related abnormalities in gross pathology, histopathology, hematology, or clinical chemistry. However, slight but statistically insignificant increases in hemoglobin concentration and erythrocyte counts were seen in the 0.01 and 0.20 ppm male rats and the 0.05 ppm female rats (USEPA, 1984).

Male and female rats chronically exposed (30 weeks) via inhalation to doses of 0, 0.05, 0.1, and 0.5 ppm HCPD 6 hours/day, 5 days/week exhibited a number of effects (USEPA, 1984). At the highest dose level, mortalities of males and females occurred. Males in this dose group exhibited depressed weight gain following the seventh week of exposure and for the remainder of the study. Females in the medium- and high-dose groups also exhibited depressed body weights. Pulmonary, kidney, and liver degenerative changes were observed in both sexes at the high dose. Kidney weights of high dose females were significantly increased.

No reproductive impairment or evidence of teratogenicity was observed in pregnant rats orally administered HCPD at doses of 3, 10 or 30 mg/kg/day during days 6-15 of gestation (USEPA, 1984). No evidence of teratogenicity was apparent in mice or rabbits orally dosed with 0, 5, 25 or 75 mg/kg/day HCPD during days 6-15 (mice) of gestation (USEPA, 1984). Fertility was not significantly different in either dosed mice or rabbits. No maternal toxicity or embryotoxicity occurred in treated mice; however, maternal toxicity did occur at 75 mg/kg/day in rabbits. No embryotoxic effects were noted at any dose level in rabbits (USEPA, 1984).

No evidence of the carcinogenicity of HCPD has been demonstrated in animals or humans (USEPA, 1984); however, the National Toxicology Program (NTP) was scheduled to start carcinogenicity testing in 1986 (NTP, 1986). It has not been shown to be mutagenic in a variety of bacterial (E. Coli, S. typhinnurium) and mammalian cell cultures (mouselymphoma).

TOXICITY TO WILDLIFE AND DOMESTIC ANIMALS

Aquatic Life

Freshwater and marine aquatic organisms exhibit acute toxic effects at concentrations of HCPD as low as 7 µg/l, while freshwater organisms exhibit chronic effects at concentrations of 5µg/l (USEPA, 1986b).

Plants

Information was not found in the literature reviewed regarding toxicity of HCPD to plants.

Invertebrates

Information was not found in the literature reviewed regarding toxicity of HCPD to invertebrates.

Birds

Information was not found in the literature reviewed regarding toxicity of HCPD to birds.

Mammals

Very little information is available on the toxicity of HCPD to wild and domestic animals. The acute oral LD_{50} of HCPD in rabbits ranges between 420 and 620 mg/kg (USEPA, 1980).

REGULATIONS AND RECOMMENDED GUIDELINES

Ambient Water Quality Criteria (USEPA, 1986b):

The available data are not adequate for establishing criteria. However, USEPA does report the lowest values known to be toxic in aquatic organisms.

Aquatic Life (Freshwater)

Acute Toxicity: 7 ug/liter
Chronic Toxicity: 5.2 ug/liter

Aquatic Life (Saltwater)

Acute Toxicity: 7 ug/liter
Chronic Toxicity: Data are insufficient

Human Health

Criterion: 206 ug/liter

OSHA PEL 29 CFR 1910.1000

TWA = 0.01 ppm

ACGIH TLV

TWA = 0.01 ppm

DOSE-RESPONSE ASSESSMENT

The human dose-response parameter estimates for carcinogens and noncarcinogens are computed differently by USEPA, therefore, these estimates are presented separately below.

Carcinogenic Effects

USEPA has not developed oral or inhalation cancer potency estimates for HCPD.

Noncarcinogenic Effects

The oral intake reference dose (RfD) for HCPD has been derived by the USEPA and is 0.007 mg/kg/day (USEPA, 1990a). The oral RfD was based on a subchronic oral (gavage) toxicity study in which male and female rats were administered HCPD of 0, 10, 19, 38, 75, or 150 mg/kg/day, 5 days/week for 13 weeks (Abdo et al., 1984). Stomach lesions were observed in 2 of 8 surviving females at 19 mg/kg/day. The inhalation intake RfD is 2 x 10^{-5} mg/kg/day (USEPA, 1990b). The RfD is based on a subchronic inhalation study in which rats were exposed to concentrations of 1.67 mg/m^3 (0.2 mg/kg/day) HCPD 5 days/week over a 13-week period (USEPA, 1990b). Though additional details of the study were not provided the toxicological effect of concern involved lesions of the respiratory tract.

Oral RfD = 0.007 mg/kg/day

Inhalation RfD = 2 x 10^{-5} mg/kg/day

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ISODRIN

SUMMARY

Data on the carcinogenicity, teratogenicity, mutagenicity, chronic toxicity, or reproductive toxicity of isodrin were not located in the literature reviewed for animals or humans. The acute oral toxicity of isodrin in young rats (90 days of age) was 7 mg/kg.

CAS Number: 465-73-6

Chemical Formula: $C_{12}H_8Cl_6$

IUPAC Name: 1,2,3,4,10,10-hexachloro-1,4,4a,5,8,8a-hexahydro-1,4,5,8-endo-endo-dimethanonaphthalene

Important Synonyms and Trade Names: Isodrin; Compound 711

CHEMICAL AND PHYSICAL PROPERTIES

Molecular Weight: 365

Melting Point: 240-242°C (Merck, 1983)

Solubility in Water: 0.16 mg/liter (Lyman et al., 1982) Estimated 1.4 mg/liter
0.02 mg/liter (Lyman et al., 1982) (Eqn. 2-3 log Kow = 4.38; 6.51)

Log Octanol/Water Partition Coefficient (Kow): 6.51 (Lyman et al., 1982) Fragment Method

Soil/Water Partition Coefficient (Koc):

5,751; 82,880 (Lyman et al., 1982) Eqn 4-8 (log Kow = 4.38; 6.51)
7,448; 339,900 (Lyman and Loreti, 1987) (log Kow = 4.38; 6.51)
8,759; 294,900 (Kadeg et al., 1986) (low Kow = 4.38; 6.51)

Bioconcentration Factor:

11,708 (Davies and Dobbs, 1984) Eqn B (log Kow = 6.5)
51,286 (Lyman et al., 1982) Eqn 5-2 (log Kow = 6.5)
4,436 (Davies and Dobbs, 1984) Eqn C (log Kow = 6.5)
1,737 (Davies and Dobbs, 1984) Eqn A (S = .16)
233 (Davies and Dobbs, 1984) Eqn C (log Kow = 4.38)
635 (Davies and Dobbs, 1984) Eqn B (log Kow = 4.38)
1,260 (Lyman et al., 1982) Eqn 5-2 (log Kow = 4.38)

Vapor Pressure: $<1 \times 10^{-4}$mm Hg [estimated for 25°C] (Cogley and Foy, 1978)

Henry's Law Constant: 4.8×10^{-4} atm-m3/mole (calculated)
3.4×10^{-5} atm-m3/mole (calculated)
1.4×10^{-3} Dimensionless
3.2×10^{-3} atm-m3/mole (calculated)
1.3×10^{-1} Dimensionless

TRANSPORT AND FATE

Very little information is available on the fate and transport of isodrin under environmental conditions; indeed, the physical/chemical properties of this compound have not yet been fully characterized. Photodrin formation has been observed following reactions of isodrin with acid, bromine, hydrogen bromide, and ultraviolet (UV) light in the laboratory (Berkowitz et al., 1978). However, under field conditions, photoconversion of isodrin to photodrin is not expected, as the maximum UV absorption of isodrin (198 nm) occurs in a region of the atmosphere where solar radiation is attenuated by both the ozone layer and by water (Berkowitz et al., 1978).

Isodrin is estimated to have a very low vapor pressure and a relatively low solubility in water (Cogley and Foy, 1978). Therefore, it appears reasonable to assume that volatilization of isodrin to air and leaching of isodrin-contaminated soil residues to groundwater will not occur to an appreciable extent. A range of Koc is reported above and indicates that sorption of isodrin to soils/sediments and dissolved organic material will occur. Pavlou (1980) estimates that sorption of chlorinated hydrocarbon pesticides is very high. The combined low estimated water solubility and high organic partitioning indicate that isodrin will exhibit little environmental mobility. The persistence of isodrin in various soils under varying experimental conditions, as summarized by Berkowitz et al. (1978), indicates that detectable residues may be present in excess of 13 years post-application.

No residues of isodrin were found in soybeans, corn, or oats grown in soil treated with isodrin (Nash et al., 1973). Ten weeks following application of isodrin (0.19 ppm) to soils, up to three percent of the applied quantity was recovered unchanged in the leaves of exposed carrots while 41 percent remained unchanged in the soils (Berkowitz et al., 1978--Cite: Klein et al.,

1973). Conversion products which accounted for a majority of the remaining residues were identified as Endrin.

A range of estimated BCFs for Isodrin is also reported above. ASTM (1985) indicates that chemicals with BCFs less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors suggests that appreciable bioconcentration or biomagnification of Isodrin residues can occur.

HEALTH EFFECTS

No information on the toxicity of isodrin to humans was located in the reviewed literature. Additionally, data on the carcinogenicity, mutagenicity, subchronic, chronic, or reproductive toxicity were not available for humans or animals in the literature reviewed. Only acute oral toxicity data are available for laboratory mice and rats. The LD_{50} values for these animals are 7-15.5 mg/kg for female and male rats (>90 days old); 16.4-27.8 mg/kg for young female and male rats (25-31 days old); and 8.8 mg/kg for mice (Berkowitz et al., 1978).

TOXICITY TO WILDLIFE AND DOMESTIC ANIMALS

Aquatic Life

Comparisons of the toxicities of isodrin and photodrin in fish to those of several other cyclodiene insecticides indicate that isodrin was more toxic (Berkowitz et al., 1978). Reported LC_{50} values for freshwater fish were 2.5, 6.0, 6.0, and 1.5 ppb in bass, bluegill, golden shiners, and goldfish, respectively (Berkowitz et al., 1978).

Plants

Information was not found in the literature reviewed regarding toxicity of isodrin to plants.

Invertebrates

Information was not found in the literature reviewed regarding toxicity of isodrin to invertebrates.

Birds

Endrin, an isomer of isodrin, was consistently the most toxic chemical among 89 chemicals tested in bobwhite, pheasants, mallards, and Japanese quail (Heath et al., 1972). Isodrin would therefore be expected to exhibit somewhat similar toxic properties.

Mammals

Limited data is available on the toxicity of isodrin in wild and domestic animals. The dermal LD_{50} in rabbits was estimated at <94 mg/kg (Berkowitz et al., 1978).

REGULATIONS AND RECOMMENDED GUIDELINES

None.

DOSE-RESPONSE ASSESSMENT

The human dose-response parameter estimates for carcinogens and noncarcinogens are computed differently by USEPA; therefore, these estimates are presented separately below.

Carcinogenic Effects

USEPA has not developed oral or inhalation cancer potency estimates for isodrin.

Noncarcinogenic Effects

The USEPA has not currently derived a reference dose (RfD) for isodrin.

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LEAD

SUMMARY

Lead is a heavy metal that exists in one of three oxidation states, 0, +2, and +4. There is suggestive evidence that some lead salts are carcinogenic, inducing kidney tumors in mice and rats. Lead is also a reproductive hazard, and it can adversely affect the brain and central nervous system by causing encephalopathy and peripheral neuropathy. Chronic exposure to low levels of lead can cause subtle learning disabilities in children. Exposure to lead can also cause kidney damage and anemia and may have adverse effects on the immune system.

CAS Number: 7439-92-1
Chemical Formula: Pb
IUPAC Name: Lead

CHEMICAL AND PHYSICAL PROPERTIES

Atomic Weight: 207.19
Boiling Point: 1,740°C
Melting Point: 327.5°C
Specific Gravity: 11.35 at 20°C (liquid)
Vapor Pressure: 1.77 mm Hg at 1,000°C (Merck, 1983)
Solubility in Water: Insoluble; some organic compounds are soluble
Solubility in Organics: Soluble in HNO_3 and hot, concentrated H_2SO_4

TRANSPORT AND FATE

Some industrially produced lead compounds are readily soluble in water (USEPA, 1979). However, metallic lead and the common lead minerals are insoluble in water. Natural compounds of lead are not usually mobile in normal surface or groundwater because the lead leached from ores is absorbed by ferric hydroxide or combines with carbonate or sulfate ions to form insoluble compounds.

Movement of lead and its inorganic and organolead compounds as particulates in the atmosphere is a major environmental transport process. Lead carried in the atmosphere can be removed by either wet or dry deposition. Although little evidence is available concerning the photolysis of lead compounds in natural waters, photolysis in the atmosphere occurs readily. These atmospheric processes are important in determining the form of lead entering aquatic and terrestrial systems.

The transport of lead in the aquatic environment is influenced by the speciation of the ion. Lead exists mainly as the divalent cation in most unpolluted waters and becomes adsorbed into particulate phases. However, in polluted waters, organic complexation is most important. Volatilization of lead compounds probably is not important in most aquatic environments.

Sorption processes appear to exert a dominant effect on the distribution of lead in the environment. Adsorption to inorganic solids, organic materials, and hydrous iron and manganese oxides usually controls the mobility of lead and results in a strong partitioning of lead to soils and the bed sediments in aquatic systems. The sorption mechanism most important in a particular system varies with geological setting, pH, Eh, availability of ligands, dissolved and particulate ion concentrations, salinity, and chemical composition. The equilibrium solubility of lead with carbonate, sulfate, and sulfide is low. Over most of the normal pH range, lead carbonate, and lead sulfate control solubility of lead in aerobic conditions, and lead sulfide and the metal control solubility in anaerobic conditions.

Lead in soil is not easily taken up by plants, and therefore its availability to terrestrial organisms is somewhat limited. Biomethylation of lead by microorganisms can remobilize lead to the environment. Bioaccumulation of lead has been demonstrated for a variety of organisms. BCFs in freshwater organisms range from 42 to 1,700 for four invertebrate and two fish species (USEPA, 1986a). In saltwater organisms, available BCFs range from 17 to 2,600 (USEPA, 1986a). Microcosm studies indicate that lead is not biomagnified through the food chain.

HEALTH EFFECTS

There is equivocal evidence that exposure to lead causes genotoxicity in humans and animals. The available evidence indicates that lead presents a hazard to reproduction and exerts a toxic effect on conception, pregnancy, and the fetus in humans and experimental animals (USEPA, 1977, 1980).

Many lead compounds are sufficiently soluble in body fluids to be toxic (USEPA, 1977, 1980). Exposure of humans or experimental animals to lead can result in toxic effects in the brain and central nervous system, the peripheral nervous system, the kidneys, and the hematopoietic system. The metabolism and retention of lead (primarily in bone) has been well studied. Chronic exposure to inorganic lead by ingestion or inhalation can cause lead encephalopathy, and severe cases can result in permanent brain damage. Lead poisoning may cause peripheral neuropathy both in adults and children. Permanent learning disabilities in children that are clinically undetectable may be caused by exposure to relatively low levels of lead. Short-term exposure to lead can cause reversible kidney damage; however, prolonged exposure at high concentrations may result in progressive kidney damage and kidney failure. Anemia, due to inhibition of hemoglobin synthesis and a reduction in the life span of circulating red blood cells, is an early manifestation of lead poisoning. Several studies with experimental animals suggest that lead may interfere with various aspects of the immune response.

Young children are deemed a high risk group for lead exposure for a number of reasons: 1) their dietary intake in mg/kg body weight is higher than that of adults; 2) young children tend to ingest greater quantities of dirt than do adults (and such soil, particularly in urban areas, can be highly contaminated); and 3) some young children have a pica habit and may consume old, lead-based paint peelings.

TOXICITY TO WILDLIFE AND DOMESTIC ANIMALS

Aquatic Life

Freshwater vertebrates and invertebrates are more sensitive to lead in soft water than in hard water (USEPA, 1980, 1983). At a hardness of about 50 mg/l $CaCO_3$, the median effect

concentrations for nine families range from 140 to 236,000 µg/l. Chronic values for Daphnia magna and the rainbow trout are 12.26 and 83.08 µg/l, respectively, at a hardness of about 50 mg/l. Acute-chronic ratios calculated for three freshwater species ranged from 18 to 62. Freshwater algae show an inhibition of growth at concentrations above 500 µg/l.

Acute values for twelve saltwater species range from 476 µg/l for the common mussel to 27,000 µg/l for the soft shell clam. The acute-chronic ratio for this species is 118. Reported BCFs range from 17.5 for the quahog clam to 2,570 for the blue mussel. Saltwater algae are adversely affected at lead concentrations as low as 15.8 µg/l.

Plants

Information was not found in the literature reviewed regarding toxicity of lead to plants.

Invertebrates

Information was not found in the literature reviewed regarding toxicity of lead to invertebrates.

Birds

Lead is known to occur in the tissue of many free-living wild animals, including birds. Reports of avian poisoning usually involve waterfowl ingesting spent lead shot with grit pebbles which they swallow to aid in digestion. Typical signs of avian lead poisoning include regurgitation, tremors, wing-droop, slowness and reluctance to move, and anorexia (Hudson et al.. 1984).

Mammals

Cases of lead poisoning have been reported for a variety of domestic animals, including cattle, horses, dogs, and cats. Several types of anthropogenic sources are cited as the source of lead in these reports. Because of their indiscriminate eating habits, cattle often experience the greater incidence of lead toxicity among domestic animals.

REGULATIONS AND RECOMMENDED GUIDELINES

Ambient Water Quality Criteria (USEPA, 1986a):

The concentrations below are for active lead, which is defined as the lead that passes through a 0.45-um membrane filter after the sample is acidified to pH 4 with nitric acid.

Aquatic Life (Freshwater)

Acute toxicity: $e^{[1.34\ (\ln(\text{hardness})) - 2.014]}$ug/liter
Chronic toxicity: $e^{[1.34\ (\ln(\text{hardness})) - 5.245]}$ug/liter

At hardness of 50, 100, and 200 mg/l $CaCO_3$ the acute criteria are 34, 82, and 200 ug/l.

At hardness of 50, 100, and 200 mg/l $CaCO_3$ the chronic criteria are 1.3, 3.2, and 7.7 ug/l.

Aquatic Life (Saltwater)

Acute toxicity: 140 ug/liter
Chronic toxicity: 5.6 ug/liter

Human Health

Criterion: 50 ug/liter

National Primary Drinking Water Standard (USEPA): 50 µg/l

NIOSH REL:

TWA = 0.10 mg/m^3 (inorganic lead)

OSHA PEL:

TWA = 0.05 mg/m^3 (inorganic lead)

ACGIH TLV

TWA = 0.15 mg/m^3 (inorganic dust and fumes)

DOSE-RESPONSE ASSESSMENT

The human dose-response parameter estimates for carcinogens and noncarcinogens are computed differently by USEPA, therefore, these estimates are presented separately below.

Carcinogenic Effects

There is evidence that several lead salts are carcinogenic in mice or rats, causing tumors of the kidneys following oral or parenteral administration. Data concerning the carcinogenicity of lead in humans are inconclusive. The available data are not sufficient to evaluate the carcinogenicity of organic lead compounds or metallic lead. Lead has been classified according to USEPA's Guidelines for Carcinogenic Risk Assessment in USEPA's Group B2 (probable human carcinogen) based upon the evidence of kidney tumors in rats following oral administration and inadequate evidence in humans (50 Federal Register 46971, Wed. Nov. 13, 1985).

Noncarcinogenic Effects

The USEPA is currently evaluating the available data to derive RfD for lead; however, these are not expected until sometime in late 1990 (USEPA, 1986c).

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MERCURY (INORGANIC)

SUMMARY

Inorganic mercury is reported to be teratogenic and embryotoxic in studies with experimental animals. In humans, prenatal exposure to mercury vapors has been associated with spontaneous abortions and infant mortalities. The major target organs for inorganic mercury compounds are the central nervous system and the kidneys. Mutagenic responses in mammalian cell cultures have been equivocal.

CAS Number: 7439-97-6

Chemical Formula: Hg

IUPAC Name: Mercury

CHEMICAL AND PHYSICAL PROPERTIES

Atomic Weight: 200.59 (Merck, 1983)

Boiling Point: 356.72°C (Merck, 1983)

Melting Point: -38.87°C (Merck, 1983)

Specific Gravity: 13.534 (Merck, 1983)

Solubility in Water: 56.2 ug/liter at 25°C (Merck, 1983)

Solubility in Organics: Depends on chemical species

Vapor Pressure: 0.0012 mm Hg at 20°C (USEPA, 1984a)
0.002 mm Hg at 25°C (Merck, 1983)

TRANSPORT AND FATE

Inorganic mercury can exist in three oxidative states in the environment, including metallic (Hg°), mercurous (Hg_2^{++}), and mercuric(Hg^{++}). In general, the mercurous salts are much less soluble than the more commonly found mercuric salts. The nature and solubility of the chemical species that occur in an environmental system will depend on the redox potential and the pH of the environment.

Mercury can volatilize to the atmosphere from aquatic and terrestrial sources. Volatilization is reduced by conversion of metallic mercury to complexed species and by deposition of HgS in reducing sediments, but even so, atmospheric transport is a major environmental distribution pathway for mercury (USEPA, 1984a). Precipitation (wet/dry) is an important mechanism for removal of mercury from the atmosphere (USEPA, 1984a). Photolysis is important in the breakdown of airborne mercurials and may be important in some aquatic systems.

Adsorption onto suspended and bed sediments is probably the most important process determining the fate of mercury in the aquatic environment. Sorption is strongest into organic material for the Hg(+2) species. Mercury in soils is generally complexed to organic compounds. Mercury is not readily leached from either organic-rich or mineral-rich soils (Rosenblatt et al., 1975). Uptake of mercury in plants can occur with the highest concentrations generally found in bulb or root crops (Rosenblatt et al., 1975). Turf grass exposed to a mixture of mercurous and mercuric chloride added to the root zone did not accumulate mercury (USEPA, 1984a). Uptake of mercury vapor by wheat leaves has been observed (USEPA, 1984a).

Virtually any mercury compound can be remobilized in aquatic systems by microbial conversion to methyl and dimethyl forms. Conditions reported to enhance biomethylation include large amounts of available mercury, large numbers of bacteria, the absence of strong complexing agents, near neutral pH, high temperatures, and moderately aerobic environments.

Inorganic mercury is bioaccumulated by numerous organisms (USEPA, 1984b). In freshwater, BCFs for mercury in mercuric chloride range from 1,800 in rainbow trout (Salmo gairdneri) to 4,994 in the fathead minnow (Pimephales promelas) (USEPA, 1984b). ASTM (1985) indicates that chemicals with BCFs less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the reported concentration factors suggests that appreciable bioconcentration or biomagnification of mercury can occur.

HEALTH EFFECTS

Occupational studies indicate that the chronic exposure to mercury vapor (Hg°) affects primarily the central nervous system and the kidneys (increased urinary excretion of high molecular weight proteins) (USEPA, 1984a). Acute exposure to high vapor concentrations can cause erethism (behavioral effects), metal fume fever, pneumonitis, bronchitis, chest pains, dyspnea, coughing, stomatitis, gingivitis, salivation, and diarrhea (USEPA, 1984a). In case reports, acute mercury vapor exposures have been shown to cause exudative alveolar and intestinal edema and erosion and desquamation and necrosis of the bronchiolar epithelium (USEPA, 1984a). Contact dermatitis may result from exposure to liquid metallic mercury (USEPA, 1984a). Soluble mercuric salts are highly toxic following ingestion as compared with the less soluble mercurous compounds.

In early studies, women chronically exposed to mercury vapor experienced increased frequencies of menstrual disturbances and spontaneous abortions (USEPA, 1984a). Rats exposed to mercury vapors (2.5 mg/m^3, 6 hr/day, 5 day/week) exhibited longer estrus cycles (USEPA, 1984a). Inorganic mercuric mercury (Hg^{++}) is translocated across the blood-brain and placental barriers to a lesser degree than Hg°, and therefore inorganic salts are less likely to affect the central nervous system and the fetus (USEPA, 1984a). Infants 4 to 30 months appear to be more susceptible than adults and older children to the effects of mercury vapors (USEPA, 1984a). Placental transport of mercury and subsequent oxidation in fetal tissues has been demonstrated in mice (USEPA, 1984a). However, no conclusive results concerning the teratogenic effects of mercury vapor are available (USEPA, 1984a). Parenteral administration of inorganic mercury salts has produced abnormalities in experimental animals (USEPA, 1984a). A number of abnormalities were reported in hamster fetuses given a subcutaneous dose of mercury acetate on day 8 of gestation, including pericardial cavity distension, cleft palate, hydrocephalus, and heart defects (USEPA, 1984a).

Mutagenic responses have been equivocal following exposure of nonmammalian cell cultures to mercuric salts in vitro (USEPA, 1984a). Chromosomal aberrations have been observed in lymphocytes of persons occupationally exposed to mercury vapors (USEPA, 1984a). Carcinogenesis in humans has not been associated with occupational exposure to mercury

vapors (USEPA, 1984a). Mercury has been classified according to USEPA's Guidelines for Carcinogenic Risk Assessment in USEPA's Group D (not classified) based upon inadequate data in animals and humans (50 Federal Register 46972, Wed. Nov. 13, 1985).

TOXICITY TO WILDLIFE AND DOMESTIC ANIMALS

Aquatic Life

The aquatic toxicity of inorganic mercury compounds has been investigated. Among freshwater species, the 96-hour LC_{50} values for inorganic mercuric salts range from 0.02 µg/l for crayfish to 2,000 µg/l for caddis fly larvae (USEPA, 1980). Mercuric chloride is acutely toxic to rainbow trout at about 300 µg/l at 10°C (USEPA, 1984a).

Plants

Information was not found in the literature reviewed regarding toxicity of mercury to plants.

Invertebrates

Information was not found in the literature reviewed regarding toxicity of mercury to invertebrates.

Birds

Chronic dietary exposure of chickens to mercuric chloride at growth inhibitory levels causes immune suppression with a differential reduction effect on specific immunoglobulins (Bridger and Thaxton, 1983). The LC_{50} values for mercuric chloride administered in the diets of Japanese quail (Coturnix c. japonica), ringed-neck pheasants (Phasianus colchicus), and mallard ducks (Aansplatyrhynchos) were 5,926, 3,790, and > 5,000 ppm, respectively (Hill et al., 1975).

Mammals

The acute oral lethal dose (low) in rabbits is 40 mg/kg (NIOSH, 1982).

REGULATIONS AND RECOMMENDED GUIDELINES

Ambient Water Quality Criteria (USEPA, 1986):

Aquatic Life (Freshwater)

Acute toxicity: 2.4 ug/liter

Chronic toxicity: 0.012 ug/liter

Aquatic Life (Saltwater)

Acute toxicity: 2.1 ug/liter

Chronic toxicity: 0.025 ug/liter

Human Health

Criterion: 144 mg/liter

National Primary Drinking Water Standard (USEPA):

0.002 mg/l

NIOSH REL:

TWA (inorganic mercury) = 0.05 mg/m^3

OSHA PEL 29 CFR 1910.1000

Ceiling Level (inorganic mercury) = 0.1 mg/m^3

ACGIH TLV

TWA (vapor) = 0.05 mg/m^3

TWA (aryl and inorganic compounds) = 0.1 mg/m^3

DOSE-RESPONSE ASSESSMENT

The human dose-response parameter estimates for carcinogens and noncarcinogens are computed differently by USEPA; therefore, these estimates are presented separately below.

Carcinogenic Effects

USEPA has not developed oral or inhalation cancer potency estimates for mercury.

Noncarcinogenic Effects

The oral intake reference dose (RfD) for mercury is 3×10^{-4} mg/kg/day reported in the Health Effects Assessment Summary Table (HEAST) (USEPA, 1990). Though details of the underlying study were not available, the effect of concern was central nervous system effects associated with blood mercury level of 200 mg/ml in exposed humans (USEPA, 1990). The inhalation intake RfD is 8.6×10^{-5} mg/kg/day (ECAO, 1990).

Oral RfD = 3×10^{-4} mg/kg/day

Inhalation RfD = 8.6×10^{-5} mg/kg/day

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METHYLENE CHLORIDE

SUMMARY

Methylene chloride is used mainly as a low temperature extractant; solvent for oils, fats, waxes, bitumen, cellulose acetate, and esters; and as a paint remover. Methylene chloride (dichloromethane) increased the incidence of lung and liver tumors and sarcomas in exposed rats and mice. Methylene chloride yielded positive results in mutagenicity tests utilizing bacterial test systems. In humans, methylene chloride irritates the eyes, mucous membranes, and skin. Exposure to high levels adversely affects the central and peripheral nervous systems, the heart, and is a chemical asphyxiant. In experimental animals, methylene chloride is reported to cause kidney and liver damage, convulsions, and paresis (incomplete paralysis).

CAS Number: 75-09-2

Chemical Formula: CH_2CL_2

IUPAC Name: Dichloromethane

Important Synonyms and Trade Names: Methylene dichloride, methane dichloride

CHEMICAL AND PHYSICAL PROPERTIES

Molecular Weight: 84.93

Boiling Point: 40°C (USEPA, 1979)

Melting Point: -95.1°C

Specific Gravity: 1.3266 at 20°C

Solubility in Water: 13,200-20,000 mg/liter at 25°C (USEPA, 1979)
19,000 mg/liter (Valvani et al., 1980)

Solubility in Organics: Miscible with alcohol and ether

Log Octanol/Water Partition Coefficient (Kow): 1.25 (USEPA, 1979)
1.30 (USEPA, 1986a)

Soil/Water Partition Coefficient (Koc):

27.5	(Sabljic, 1984) (experimental)
114; 121	(Lyman et al., 1982) Eqn 4-8 (log Kow = 1.25;1.30)
27; 30	(Lyman and Loreti, 1987) (log Kow = 1.25; 1.30)
8.8	(USEPA, 1986a)

Bioconcentration Factor (BCF):

2.9 - 2.3	(Davies and Dobbs, 1984) Eqn A (S = 13,200 - 20,000)
5.25	(Lyman et al., 1982) Eqn 5-2 (log Kow = 1.25)
8.60	(Davies and Dobbs, 1984) Eqn C (log Kow = 1.25)
5.81	(Davies and Dobbs, 1984) Eqn B (log Kow = 1.25)
16.4	(Lyman et al., 1982) Eqn 5-2 (log Kow = 1.9)
21	(Davies and Dobbs, 1984) Eqn B (log Kow = 1.9)
14.2	(Davies and Dobbs, 1984) Eqn C (log Kow) = 1.9)

Vapor Pressure: 362 mm Hg at 20°C (USEPA, 1986a)
436 mm Hg at 25°C (Berkowitz et al., 1978)

Vapor Density: 2.93

Henry's Law Constant:

2.6×10^{-3} atm-m^3/mole (Calculated)
2.03×10^{-3} atm-m^3/mole (USEPA, 1986a)
8.53×10^{-2} Dimensionless

TRANSPORT AND FATE

Volatilization to the atmosphere appears to be the major mechanism for removal of methylene chloride from aquatic systems and its primary environmental transport process (USEPA, 1979). Photooxidation in the troposphere appears to be the dominant chemical fate of methylene chloride following its release to the air. Once in the troposphere, the compound is attacked by hydroxyl radicals, resulting in the formation of carbon dioxide, and to a lesser extent, carbon monoxide and phosgene. Phosgene is readily hydrolyzed to HCl and CO_2. About one percent of tropospheric methylene chloride would be expected to reach the stratosphere where it would probably undergo photodissociation resulting from interaction with high energy ultraviolet radiation. Aerial transport of methylene chloride is partly responsible for its relatively wide environmental distribution. Atmospheric methylene chloride may be returned to the earth in precipitation.

Photolysis, oxidation, and hydrolysis do not appear to be significant environmental fate processes for methylene chloride. A range of experimental and estimated Koc is reported above and indicates that some sorption of methylene chloride to soils/sediments and dissolved organic material will occur. Pavlou (1980) estimates that sorption of volatile organic compounds will range from low to moderate. The combined high water solubility and low

organic partitioning of methylene chloride suggest that this compound will exhibit a high degree of environmental mobility. Although methylene chloride is potentially biodegradable, especially by acclimatized microorganisms, biodegradation occurs at a very slow rate.

A range of BCFs for methylene chloride is also reported above. ASTM (1985) indicates that chemicals with BCFs less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors suggest that appreciable bioconcentration or biomagnification of methylene chloride residues is not likely to occur.

HEALTH EFFECTS

Results of a National Toxicology Program Study on methylene chloride (USEPA, 1990) indicate increased incidence of lung and liver tumors in mice and mammary tumors in female and male rats exposed to methylene chloride. In a chronic inhalation study, male rats exhibited an increased incidence of sarcomas in the ventral neck region (Burek et al., 1984); however, the authors suggest that the relevance and toxicological significance of this finding is uncertain in light of available toxicity data. Methylene chloride has been classified according to USEPA's Guidelines for Carcinogenic Risk Assessment, in USEPA's Group B2 (probable human carcinogen), based upon positive results in animal studies and inadequate evidence in humans (USEPA, 1985b).

Methylene chloride is reported to be mutagenic in bacterial test systems. It has also produced positive results in the Fischer rat embryo cell transformation test. However, it has been suggested that the observed cell-transforming capability may have been due to impurities in the test material. There is no conclusive evidence that methylene chloride exposure produces teratogenic effects.

In humans, direct contact with methylene chloride produces eye, respiratory tract, and skin irritation (USEPA, 1985b). Mild poisonings due to inhalation exposure produce somnolence, lassitude, numbness and tingling of the limbs, anorexia, and light headedness, followed by rapid and complete recovery. More severe poisonings generally involve correspondingly greater disturbances of the central and peripheral nervous systems. Methylene chloride also

has acute toxic effects on the heart, including the induction of arrhythmia. Fatalities reportedly due to methylene chloride exposure have been attributed to cardiac injury and heart failure. Methylene chloride is metabolized to carbon monoxide in vivo, and levels of carboxyhemoglobin in the blood are elevated following acute exposures. In experimental animals, methylene chloride is reported to cause kidney and liver damage, convulsions, and distal paresis. An oral LD_{50} value of 2,136 mg/kg, and an inhalation LC_{50} value of 88,000 mg/m^3/30 minutes are reported for the rat.

TOXICITY TO WILDLIFE AND DOMESTIC ANIMALS

Aquatic Organisms

Very little information concerning the toxicity of methylene chloride to domestic animals and wildlife exists (USEPA, 1980). Acute values for the freshwater species Daphnia magna, the fathead minnow, and bluegill are 224,000, 193,000, and 224,000 µg/liter, respectively. Acute values for the saltwater mysid shrimp and sheepshead minnow are 256,000 and 331,000 µg/liter, respectively. No data concerning chronic toxicity are available. The 96-hour EC_{50} values for both freshwater and saltwater algae are greater than the highest test concentration, 662,000 µg/liter.

Plants

Information was not found in the literature reviewed regarding the toxicity of methylene chloride to plants.

Invertebrates

Information was not found in the literature reviewed regarding the toxicity of methylene chloride to invertebrates.

Birds

Information was not found in the literature reviewed regarding the toxicity of methylene chloride to birds.

Mammals

Information was not found in the literature reviewed regarding the toxicity of methylene chloride to mammals.

REGULATIONS AND RECOMMENDED GUIDELINES

Ambient Water Quality Criteria (USEPA, 1986b):

Available data are not adequate for establishing criteria, however, USEPA does report the lowest values known to be toxic in aquatic organisms:

Aquatic Life (Freshwater)

Acute toxicity: 11,000 µg/liter

Chronic toxicity: No data are available

Aquatic Life (Saltwater)

Acute toxicity: 12,000 µg/liter

Chronic toxicity: 6,400 µg/liter

Human Health

Due to the carcinogenicity of methylene chloride the ambient water criterion is set at zero. However, estimates of the carcinogenic risks associated with lifetime exposure from ingestion of contaminated water and contaminated aquatic organisms are:

Risk	Concentration
10^{-5}	1.9 µg/liter
10^{-6}	0.19 µg/liter
10^{-7}	0.019 µg/liter

OSHA PEL 29 CFR 1910.1000:

8-Hour Time-Weighted Average (TWA) = 100 ppm

Ceiling Level = 200 ppm

Peak Concentration = 300 ppm (5 minutes in any 3 hours)

NIOSH REL:

TWA = 75 ppm

ACGIH TLV:

TWA = 50 ppm (suspect human carcinogen)

DOSE-RESPONSE ASSESSMENT

The human dose-response parameter estimates for carcinogens and noncarcinogens are computed differently by USEPA; therefore, these estimates are presented separately below.

Carcinogenic Effects

The Cancer Assessment Group (CAG) of the USEPA has derived an oral cancer potency estimate for methylene chloride of 7.5×10^{-3} $(mg/kg/day)^{-1}$ (USEPA, 1989). This estimate is based on the arithmetic mean of the potency slope factors determined for hepatocellular adenomas and carcinomas in mice derived from the lifetime inhalation exposure studies conducted by the National Toxicology Program (NTP, 1986) and the National Coffee Association (NCA, 1983). The CAG has also derived an inhalation cancer potency estimate of 1.4×10^{-2} $(mg/kg/day)^{-1}$ based on the combined incidence of adenomas and carcinomas of the liver or lung from the NTP study (NTP, 1986).

Oral Cancer Potency Estimate: 7.5×10^{-3} $(mg/kg/day)^{-1}$ (USEPA, 1989)
Inhalation Cancer Potency Estimate: 1.4×10^{-2} $(mg/kg/day)^{-1}$ (USEPA, 1989)

Noncarcinogenic Effects

The USEPA has computed a chronic oral reference dose (RfD) of 6×10^{-2} mg/kg/day for methylene chloride (USEPA, 1989) based on a 2-year drinking water study with rats, which identified a NOAEL (no-observed-adverse-effect-level) of 6 mg/kg/day (NCA, 1982). Higher doses produced histological alterations of the liver. An uncertainty factor of 100 was incorporated to account for uncertainties in extrapolating animal data to humans (10) and to account for sensitive human subgroups (10) (USEPA, 1989). An inhalation RfD is not currently available.

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TETRACHLOROETHYLENE

SUMMARY

Tetrachloroethylene (PCE) induced liver tumors following oral administration to mice. PCE has also been shown to be mutagenic in bacterial systems. Reproductive toxicity was observed in pregnant rats and mice following exposure to high concentrations of PCE. Animals exposed via inhalation exhibited liver, kidney, and central nervous system damage. In humans, central nervous system depression and liver toxicity are also the principal effects exhibited following PCE exposure.

CAS Number: 127-18-4

Chemical Formula: C_2Cl_4

IUPAC Name: Tetrachloroethene

Important Synonyms and Trade Names: Perchloroethylene, PCE

CHEMICAL AND PHYSICAL PROPERTIES

Molecular Weight: 165.83

Boiling Point: 121°C

Melting Point: -22.7°C

Specific Gravity: 1.63

Solubility in Water: 150 - 200 mg/liter at 20°C

Solubility in Organics: Soluble in alcohol, ether, and benzene

Log Octanol/Water Partition Coefficient (Kow): 2.60 (Hansch and Leo, 1979)
2.53 (Veith et al., 1983)

Soil/Water Partition Coefficient (Koc):

270; 306 (Lyman and Loreti, 1987) (log Kow = 2.53; 2.60)
360 (Chiou et al., 1979) (experimental)
567; 619 (Lyman et al., 1982) Eqn 4-8 (log Kow = 2.53, 2.6)
364 (USEPA, 1986a)

Bioconcentration Factor:

49 (Davies and Dobbs, 1984) (Table 2) (experimental)
38-19 (Davies and Dobbs, 1984) Eqn A (S = 140-500)
30.6 (USEPA, 1980)
55.7 (Lyman et al., 1982) Eqn 5-2 (log Kow = 2.6)
49.3 (Lyman et al., 1982) Eqn 5-2 (log Kow = 2.53)
26.9 (Davies and Dobbs, 1984) Eqn C (log Kow = 2.55)
51.3 (Davies and Dobbs, 1984) Eqn B (log Kow = 2.55)
51.1 (Lyman et al., 1982) Eqn 5-2 (log Kow = 2.55)

Vapor Pressure: 14 mm Hg at 20°C
17.8 mm Hg (USEPA, 1986a)

Henry's Law Constant: 1.4×10^{-2} atm-m^3/mole (calculated)
2.59×10^{-2} atm-m^3/mole (USEPA, 1986a)
1.09 Dimensionless

TRANSPORT AND FATE

PCE volatilizes rapidly into the atmosphere where it reacts with hydroxyl radicals to produce HCl, CO, CO_2, and carboxylic acid. This is probably the most important transport and fate process for PCE in the environment. The half-life of PCE in air is approximately 47 days (USEPA, 1984). The half-life of PCE in water may range from 1-30 days (USEPA, 1986a).

The range of experimental and estimated Kocs reported above indicates that sorption of PCE to soils/sediments and dissolved organic material will occur. Pavlou (1980) estimates that sorption of volatile organic compounds will range from low to moderate. The combined water solubility and organic partitioning data indicate that PCE will exhibit some degree of environmental mobility. It is uncertain if organically bound PCE can be efficiently degraded by microorganisms.

A range of experimental and estimated BCFs for PCE is also reported above. ASTM (1985) indicates that chemicals with BCFs less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors suggests that appreciable bioconcentration or biomagnification of PCE residues is not likely to occur.

HEALTH EFFECTS

Health effects in humans following chronic exposure to PCE include respiratory tract irritation, nausea, headache, sleeplessness, abdominal pains, constipation, liver cirrhosis, hepatitis, and nephritis (USEPA, 1984). However, central nervous system depression and liver toxicity are the principal systemic effects exhibited following PCE exposure (acute, chronic). Blair et al. (1979) observed an excess of lung, cervical, and skin cancers and slight increases in leukemia and liver cancer in a study of deceased laundry and dry-cleaning workers with known exposures to PCE, carbon tetrachloride, and trichloroethylene.

PCE was not mutagenic in several Salmonella typhimurium strains either with or without metabolic activation (NTP, 1986). It was not mutagenic in mouse lymphoma cells with or without metabolic activation and did not induce sex-linked recessive lethal mutations in Drosophila melanogaster (NTP, 1986). PCE did not induce sister chromatid exchanges or chromosomal aberrations in Chinese hamster ovary cells, with or without metabolic activation.

In male and female mice, PCE was found to produce liver cancer when orally administered by gavage (NCI, 1977). The NTP recently completed a chronic (103 week) inhalation study with PCE in rats and mice (NTP, 1986). The exposure concentrations were 0, 200, or 400 ppm for rats and 0, 100, or 200 ppm for mice. Survival of male rats was affected at the high dose, and survival of male mice was affected at both doses. Survival of female mice was reduced at 200 ppm. Both concentrations of PCE were associated with leukemia in male and female rats. PCE caused renal tubular cell hyperplasia in male rats, renal tubular cell adenomas or adenocarcinomas in male rats (not statistically significant), and renal tubular cell karyomegaly in male and female rats. One low-dose male rat had a kidney lipoma and another had a nephroblastoma. In male and female mice, PCE caused increased incidences of hepatocellular neoplasms. High-dose males had increased incidences of hepatocellular adenomas, while an increased incidence of hepatocellular carcinomas occurred at both concentrations in males and females.

As was observed in rats, PCE produced renal tubular cell karyomegaly. No neoplastic changes were observed in the respiratory tracts of either species; however, an increased incidence of squamous metaplasia was observed in the nasal cavities of dosed male rats.

Delayed ossification of skull bones and sternebrae were reported in the offspring of pregnant mice exposed via inhalation to concentrations of 2,000 mg/m^3 PCE. The exposure duration was 7 hours/day and spanned days 6-15 of gestation. In another study, increased fetal resorptions were observed following exposure of pregnant rats to PCE. Renal toxicity and hepatotoxicity were exhibited by rats following chronic inhalation exposure at levels of 1,356 mg/m^3 PCE. During the first 2 weeks of a subchronic inhalation study, exposure to concentrations of 1,622 ppm (10,867 mg/m^3) of tetrachloroethylene produced signs of central nervous system depression and cholinergic stimulation in rabbits, monkeys, rats, and guinea pigs.

TOXICITY TO WILDLIFE AND DOMESTIC ANIMALS

Aquatic Life

PCE is the most toxic of the chloroethylenes to aquatic organisms. Limited acute toxicity data are available for PCE; however, these data appear to indicate that the LC_{50} values for saltwater and freshwater species are similar-approximately 10,000 µg/l. The trout was the most sensitive species evaluated (LC_{50} = 4,800 µg/l). Chronic values were 840 and 450 µg/l for freshwater and saltwater species, respectively. An acute-chronic ratio of 19 has been computed for PCE.

Plants

Information was not found in the literature reviewed regarding toxicity of PCE to plants.

Invertebrates

Information was not found in the literature reviewed regarding toxicity of PCE to invertebrates.

Birds

Information was not found in the literature reviewed regarding toxicity of PCE to birds.

Mammals

Information was not found in the literature reviewed regarding toxicity of PCE to mammals.

REGULATIONS AND RECOMMENDED GUIDELINES

Ambient Water Quality Criteria (USEPA, 1986c):

The available data are not adequate for establishing criteria. However, USEPA does report the lowest values known to be toxic to aquatic organisms.

Aquatic Life (Freshwater)

Acute toxicity: 5,280 ug/liter
Chronic toxicity: 840 ug/liter

Aquatic Life (Saltwater)

Acute toxicity: 10,200 ug/liter
Chronic toxicity: 450 ug/liter

Human Health

Due to the carcinogenicity of PCE the ambient water criterion is set at zero. However, estimates of the carcinogenic risks associated with lifetime exposure from ingestion of contaminated water and contaminated aquatic organisms are:

Risk	Concentration
10^{-4}	80 ug/liter
10^{-5}	8.0 ug/liter
10^{-6}	0.8 ug/liter
10^{-7}	0.08 ug/liter

NIOSH REL:

TWA = 100 ppm

Ceiling Level = 200 ppm

OSHA PEL 29 CFR 1910.1000

TWA = 25 ppm

ACGIH TLV

TWA = 50 ppm (suspect human carcinogen)

STEL = 200 ppm

DOSE-RESPONSE ASSESSMENT

The human dose-response parameter estimates for carcinogens and noncarcinogens are computed differently by USEPA; therefore, these estimates are presented separately below.

Carcinogenic Effects

The Cancer Assessment Group (CAG) of USEPA has derived an oral cancer potency estimate for PCE of 5.1×10^{-2} $(mg/kg/day)^{-1}$ (USEPA, 1990).

The CAG has also derived an inhalation cancer potency estimate of 3.3×10^{-3} $(mg/kg/day)^{-1}$ (USEPA, 1990).

Oral Cancer Potency Estimate: 5.1×10^{-2} $(mg/kg/day)^{-1}$ (USEPA, 1990).
Inhalation Cancer Potency Estimate: 3.3×10^{-3} $(mg/kg/day)^{-1}$ (USEPA, 1990).

Noncarcinogenic Effects

The USEPA has not currently derived a reference dose (RfD) for PCE.

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ZINC

SUMMARY

Ingestion of excessive amounts of zinc can cause fever, vomiting, and stomach cramps. Exposure to high concentrations of zinc oxide fumes can cause metal fume fever. Inhalation of mists or fumes may irritate the respiratory tract, and contact with zinc chloride may irritate the eyes and skin. High levels of zinc in the diet have been shown to retard growth and produce defective mineralization of bone. Zinc generally exists in nature as a salt with a valence of +2.

CAS Number: 7440-66-6
Chemical Formula: Zn
IUPAC Name: Zinc

CHEMICAL AND PHYSICAL PROPERTIES:

Atomic Weight: 65.38
Boiling Point: 907°C
Melting Point: 419.58°C
Specific Gravity: 7.133 at 25°C
Solubility in Water: Insoluble; some salts are soluble
Solubility in Organics: Soluble in acid and alkali
Vapor Pressure: 1 mm Hg at 487°C

TRANSPORT AND FATE

Zinc can occur in both suspended and dissolved forms. Dissolved zinc may occur as the free (hydrated) zinc ion or as dissolved complexes and compounds with varying degrees of stability and toxicity. Suspended (undissolved) zinc may be dissolved following minor changes in water chemistry or may be sorbed to suspended matter. The predominant fate of zinc in aerobic aquatic systems is sorption of the divalent cation by hydrous iron and manganese oxides, clay minerals, and organic material. The efficiency of these materials in removing zinc from solution varies according to their compositions and concentrations, the pH and salinity of the water, the concentrations of complexing ligands, and the concentration of zinc.

Concentrations of zinc in suspended and bed sediments always exceed concentrations in ambient water. In reducing environments, precipitation of zinc sulfide limits the mobility of zinc. However, under aerobic conditions, precipitation of zinc compounds is probably important only where zinc is present in high concentrations. Zinc tends to be more readily sorbed at higher pH than lower pH and tends to be desorbed from sediments as salinity increases. Compounds of zinc with the common ligands of surface waters are soluble in most neutral and acidic solutions, so that zinc is readily transported in most unpolluted, relatively organic-free waters.

The relative mobility of zinc in soil is determined by the same factors affecting its transportation in aquatic systems. Atmospheric transport of zinc is also possible. However, except near sources such as smelters, zinc concentrations in air are relatively low and fairly constant.

Since it is an essential nutrient, zinc is bioaccumulated even in the absence of abnormally high ambient concentrations. Zinc does not appear to be biomagnified. Although zinc is actively bioaccumulated in aquatic systems, the biota appear to represent a relatively minor sink compared to the sediments. Zinc is one of the most important metals in biological systems. Since it is actively bioaccumulated, the environmental concentrations of zinc probably exhibit seasonal fluctuations.

HEALTH EFFECTS

Testicular tumors have been produced in rats and chickens when zinc salts are injected intratesticularly, but not when other routes of administration are used (USEPA, 1984). Zinc may be indirectly important with regard to cancer since its presence seems to be necessary for the growth of tumors. Laboratory studies suggest that although zinc-deficient animals may be more susceptible to chemical induction of cancer, tumor growth is slower in these animals (USEPA, 1984). There is no evidence that zinc deficiency has any etiological role in human cancer. There are no data available to suggest that zinc is mutagenic or teratogenic in animals or humans (USEPA, 1984).

Zinc is an essential trace element that is involved in enzyme functions, protein synthesis, and carbohydrate metabolism (USEPA, 1984). Ingestion of excessive amounts of zinc may cause fever, vomiting, stomach cramps, and diarrhea. Zinc oxide fume can penetrate deep into the alveoli and cause metal fume fever (USEPA, 1984). Zinc oxide dust does not produce this disorder. Contact with zinc chloride can cause skin and eye irritation. Inhalation of mists or fumes may irritate the respiratory and gastrointestinal tracts. Zinc in excess of 0.25 percent in the diet of rats causes retardation, hypochromic anemia, and defective mineralization of bone (USEPA, 1984). No zinc toxicity is observed at dietary levels below 0.25 percent.

Studies with animals and humans indicate that metabolic changes may occur due to the interaction of zinc and other metals in the diet. Exposure to cadmium can cause changes in the distribution of zinc, with increases in the liver and kidneys (organs where cadmium also accumulates). Excessive intake of zinc may cause copper deficiencies and result in anemia. Interaction of zinc with iron or lead may also lead to changes that are not produced when the metals are ingested individually. USEPA has classified zinc in Group D (not classifiable) according to the Guidelines for Carcinogenic Risk Assessment.

TOXICITY TO WILDLIFE AND DOMESTIC ANIMALS

Aquatic Life

Zinc produces acute toxicity in freshwater organisms over a range of concentrations from 90 to 58,100 µg/l and appears to be less toxic in harder water (USEPA, 1980). Acute toxicity is similar for freshwater fish and invertebrates (USEPA, 1980). Chronic toxicity values range from 47 to 852 µg/l and appear to be relatively unaffected by hardness (USEPA, 1980). A final acute-chronic ratio for freshwater species of 3.0 has been reported. Although most freshwater plants appear to be insensitive to zinc, one species, the alga Selenastrum capricornutum, exhibited toxic effects at concentrations from 30 to 700 µg/l (USEPA, 1980). Reported acute toxicity values range from 2,730 to 83,000 µg/l for saltwater fish and from 166 to 55,000 µg/l for invertebrate saltwater species (USEPA, 1980). Zinc produces a chronic toxicity in the mysid shrimp at 166 µg/l. The final acute-chronic ratio for saltwater species is 3.0. Toxic effects are observed in saltwater plant species at zinc concentrations of 50 to 25,000 µg/l (USEPA, 1980).

Plants

Information was not found in the literature reviewed regarding toxicity of zinc to plants.

Invertebrates

Information was not found in the literature reviewed regarding toxicity of zinc to invertebrates.

Birds

Information was not found in the literature reviewed regarding toxicity of zinc to birds.

Mammals

Zinc poisoning has occurred in cattle. In one outbreak, poisoning was caused by food accidentally contaminated with zinc at a concentration of 20 g/kg. An estimated intake of 140 g of zinc per cow per day for about 2 days was reported. The exposed cows exhibited severe arthritis, and some died or had to be slaughtered. Postmortem findings showed severe pulmonary emphysema with changes in the myocardium, kidneys, and liver. Zinc concentrations in the liver were extremely high. Based on relatively limited data, some researchers have speculated that exposure to excessive amounts of zinc may constitute a hazard to horses. Laboratory studies and findings in foals living near lead-zinc smelters suggest that excessive exposure to zinc may produce bone changes, joint afflictions, and lameness. In pigs given dietary zinc at concentrations greater than 1,000 mg/kg, decreased food intake and weight gain were observed. At dietary levels greater than 2,000 mg/kg, deaths occurred as soon as 2 weeks after exposure; severe gastrointestinal changes and brain damage, both of which were accompanied by hemorrhages, were observed, as well as changes in the joints. High concentrations of zinc were found in the liver in these same studies.

REGULATIONS AND RECOMMENDED GUIDELINES

Ambient Water Quality Criteria (USEPA, 1986):

Aquatic Life (Freshwater)

Acute toxicity: $e^{[0.83(\ln(\text{hardness})) + 1.95]}$ug/liter
Chronic toxicity: 47 ug/liter

At hardness of 50, 100, and 200 mg/liter $CaCO_3$, the acute criteria are 180, 320, and 570 ug/liter.

Aquatic Life (Saltwater)

Acute toxicity: 170 ug/liter
Chronic toxicity: 58 ug/liter

Human Health

Organoleptic criterion: 5 mg/liter

NIOSH REL:

5 mg/m^3 (zinc oxide)

OSHA PEL: 29 CFR 1910.1000

TWA = 10 mg/m^3 (zinc oxide total dust)
= 5 mg/m^3 (zinc oxide respirable fraction)

ACGIH TLV

Zinc chloride fume: TWA = 1 mg/m^3
STEL = 2 mg/m^3

Zinc oxide fume: TWA = 5 mg/m^3
STEL = 10 mg/m^3

Zinc oxide dust: TWA = 10 mg/m^3 (nuisance particulate)

DOSE-RESPONSE ASSESSMENT

The human dose-response parameter estimates for carcinogens and noncarginogens are computed differently by USEPA; therefore, these estimates are presented separately below.

Carcinogenic Effects

USEPA has not developed oral or inhalation cancer potency estimates for zinc.

Noncarcinogenic Effects

The oral intake reference dose (RfD) for zinc is 0.2 mg/kg/day based on therapeutic dosages (2.14 mg/kg/day) to humans (USEPA, 1990). The inhalation intake RfD has not been determined for zinc at this time.

Oral RfD = 0.2 mg/kg/day

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